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DUST AND HISTORY.[1]

By DOUGLAS GORDON,

Director of Industrial Medicine, Department of Public Health of Queensland, Brisbane.

Introduction.

My story of pulmonary dust disease must necessarily start in prehistoric mists, for it is in some ways a story of mining—an industry which started when man first ceased to use his bare hands and wooden weapons and looked for tools (the material for which he found in the earth), and later developed as his social organization became more complex, to supply metals for his currency, utensils for his hearth, and weapons for his wars, and finally reached its zenith in our technological civilization. Practically every facet of our existence basically depends on the materials taken from the ground to supply power and be made into machines used by our primary and secondary industries to produce and process our foodstuffs and to fashion the articles which we use in our daily lives. Even power produced by water depends on machines made from ores taken from the deeps of the earth. The atomic age will be no exception. I make no apology for this, for the history of an occupational disease is essentially linked with the industrial, social and economic history of the industries which cause it.

[1] Presidential address, delivered at the inaugural clinical meeting of the Thoracic Society of Queensland.

Miners of the Mists.

Palæolithic man sought from the earth flint, ochre and salt, and his neolithic successor developed the search for flint into an important industry, for freshly extracted flint was more easily fashioned than flint long exposed to the weather. All over Europe remains of these mines have been found, and skeletons have been discovered in them of men killed by falls of earth. (Bony deformities in one such skeleton have been ascribed to poliomyelitis.) These mines had shafts and primitive tunnels and were lighted by crude lamps.

In pulmonary tissues of this period evidence of carbonization and silicosis with pleural adhesions were observed by Wilder and Wilson. Collis noted in 1915 that the flint knappers of Brandon were still using tools similar in shape to the prehistoric deerhorn picks of their olden trade ancestors. We can therefore at least wonder whether the flint workers of old suffered the pulmonary ravages so common in their occupational descendants of modern times.

Slavery and Mining of Ancient History.

Much of ancient history as we know it was cradled in the Mediterranean—first Egypt, then Greece, and finally Rome. The Egyptians sought in the barren Sinai peninsula copper for tools, weapons and utensils, and turquoise, lapis lazuli and malachite for jewellery. Much of this was found in sandstone. Nubia and the granite mountains of the Red Sea region provided gold. At first the mining was carried out by "grab and run" expeditions, which carried

all supplies, including water. One such consisted of 754 men. Later permanent mining colonies were established. The mining engineer of today, seeking his riches in the arid and uninhabited wastes of the earth, would sympathize with the difficulties the Egyptians encountered with primitive transport and poor lines of communication. Times do not change greatly. Much of our own metalliferous mining is carried on in inaccessible places. Considering that Wood Jones and others observed pleural adhesions in Egyptian mummies and Ruffer anthracosis, and considering that the natural rock was sandstone and granite, we can again wonder about the existence of dust diseases of the lungs.

Unfortunately, no one bothered to record much about these miners, for they were slaves, and I would think it miraculous if any lived long enough to develop silicosis.

Rosen quotes Diodorus (about 50 B.C.) as follows:

> . . . the kings of Egypt collect condemned prisoners, prisoners of war and others who, beset by false accusations, have been, in a fit of anger thrown into prison; these sometimes alone, sometimes with their entire families, they send to the gold mines; partly to exact a just vengeance for crimes committed by the condemned, partly to secure for themselves a big revenue through their toil. Those who have been thus consigned are many, and all are fettered; they are held constantly at work by day and the whole night long without any rest, and are sedulously kept from any chance of escape. For their guards are foreign soldiers, all speaking different languages, so the workers are unable either by speech or by friendly entreaty to corrupt those who watch them. . . . As these workers can take no care of their bodies and have not even a garment to hide their nakedness, there is no one, who seeing these luckless people would not pity them because of the excess of their misery, for there is no forgiveness or relaxation at all for the sick, or the maimed, or the old, or for women's weakness, but all with blows, are compelled to stick to their labour until worn out they die in servitude. Thus the poor wretches ever account the future more dreadful than the present because of the excess of their punishment and look to death as more desirable than life.

These conditions were apparently typical of all miners of antiquity. As a matter of fact, at a later date the more enlightened of the Romans considered that ordinary slaves should not be sent to the mines, where the conditions were completely intolerable and where the expectancy of life was but fleeting, and that such labours should be reserved for criminals and prisoners of war.

That part of our philosophy and that part of our civilization which we have inherited from Greece depends perhaps on the fact that relatively barren Attica possessed unsurpassed riches in the silver mines of Laureon. This enabled the Athenians to buy much of their foodstuffs and devote much of their energy to the arts and philosophical dissertation, and also to their innumerable intrigues, schemes, alliances and wars. The mines were worked by slaves, and at times their masters had little luck with them. There were some fearsome and bloody revolts. Fortunately the mines were dry, so drainage difficulties did not arise, but the problems of ventilation were seldom handled adequately, and apparently the air in most workings was intolerable.

At a later date still Iberia (Spain) was the real El Dorado of the ancient world. Rome and Carthage fought each other to decide—among other things—which should own her riches, for her earth abounded in copper, silver and gold. Here all the problems—drainage and ventilation—and horrors of ancient mining were revived on a huge scale. The miner was a creature half-way between man and beast, who suffered greatly while he lived and anyway lived only but a short time. It was taken for granted that he would be unhealthy. Water in the mines was an acute problem which the appearance of the Archimedean screw pump did not really solve. Ventilation was poor; the furnace atmosphere was made worse by the use of fire setting to split rock; hookworm flourished with epidemic and fatal anæmia; accidents were frequent, and the lash was ever present. If dust disease existed—and it may not have, for the mines were so wet—it would have been almost a pleasing release. This much at least could be said for the Romans—they developed timbering in mines, they had a sketchy knowledge of underground gases and did devise a rough safety code, and, as will be seen later, they did not have a monopoly of mining horror.

The "Bergsucht" (Miners' Sickness).

It is an historian's privilege to cross the centuries as though they were not, and so we come in the tenth century to Central Europe and more particularly to Bohemia, Saxony and Silesia, the Tyrol and Carinthia. With the decline of Rome the blanket of the dark descended on Europe, and though mining was carried on in an intermittent manner under a form of serfdom, we know little of it. Silver and lead were at first the prizes, but other substances such as iron, copper, sulphur, bismuth and arsenic were sought. The area was a veritable treasure chest; but the presence of lead, mercury, arsenic and radioactive dusts—in the light of present knowledge—obviously complicated the medical problem. As the spirit of civilization once again crept hesitantly forth from the ruins of the centuries, metals became more and more in demand and the area underwent progressive development. Improvements in metallurgy greatly assisted this. It was not until the rich mines of the New World enriched the old that German mining began to decline in the sixteenth and seventeenth centuries and practically disappeared in the eighteenth. The devastating effects of the religious wars also helped this process.

Socially the miners of these centuries began as serfs, but became so skilled and important that they were made free men with their own guilds, mines on tribute, cities and codes of safety, and then eventually as capital was needed to work the deepening mines and mining became more complex, they had by the sixteenth century regressed to a depressed class more commonly known today as the proletariat. In the middle centuries of the Middle Ages the standard of living for all was undoubtedly primitive and raw, but at no other time have miners been possessed of such working freedom and such a relatively high standard of living compared with the norm of their times. In 1524 the miners were crushed with the peasants in the Peasants' War and went out of history as an integral body for three centuries.

Against this background from the tenth century onwards there had grown up a very definite tradition of a disease entity from which miners suffered, known as the "*Bergsucht*". Until the beginning of the sixteenth century little knowledge of it was preserved in writing. From then onwards, however, there was a spate of literature on the subject, and most of the better known German medical writers of those times had something to say about it. In the beginning, as a clinical entity it was obviously a "hotpotch" of silicosis, pulmonary neoplasms, arsenical and mercurial poisoning and ancylostomiasis. The pathogenesis had its roots in demonology (small demons were said to be encountered from time to time in the mines), in alchemy, in "humours" and in one or two sound observations. Agricola ("*De re metallica*", 1556) knew that dust caused a pulmonary lesion, and recommended respirators. This wasting disease due to a lung lesion was apparently well known in some regions. He says:

> In the Mines of the Carpathian Mountains women are found who have married seven husbands, all of whom this terrible consumption has carried off to a premature death.

By 1652 Ursinus in "*De morbis metalloriorum*" had arrived at a clinical entity—a long drawn out "peripneumonia" due to inhaling dust—and Stockhausen ("*Appendix ueber die Bergsucht*", 1656) recommended the avoidance of dust and fumes. The clinical disease entity of his time was breathlessness with a short hard cough which later might go on to "consumption".

Hoffman, in 1738, described the Schneeberg and Joachimstal lung cancers; Henkel in 1745 was trying to measure dust concentrations in mines; and Scheffler (1770) knew that there was an early "asthmatic" stage of the

disease associated with emphysema of the lung, and that to this a secondary infection might be added later.

By this time German mining had declined, but not before Casper Weindl, a Tyrolean, had introduced gunpowder for underground blasting in 1627, and Hutmann (1636) had tried out a crude drilling machine. These "innovations" have kept us in plenty of trouble with dust ever since.

"The Black Spit".

"The black spit" was the problem of the first half of the nineteenth century. The steam age paved the way for the industrialization of England, and for steam large quantities of coal were needed. British industrialization has been summed up as "a laying of population and enterprise upon the areas which had coal underneath". Coal mining had existed in England since 1217 at least, but it was in the nineteenth century that really rapid expansion of the industry took place. In 1700 the annual production of coal was 2,612,000 tons; in 1800 it was 10,000,000 tons; in 1851 it was 57,000,000 tons; in 1897 it was 200,000,000 tons. (Queensland production in 1950 was 2,250,000 tons.)

At the beginning of the nineteenth century pits were in the main small, shallow and scattered, though one mine was down to 993 feet. (The deepest shaft in Queensland at present is about 700 feet.) The drainage problem had been greatly helped by Thomas Savery's fire engine for pumping; this was in use during the eighteenth century, but transport underground, haulage to the surface and particularly mine ventilation were gnawing problems. Poor air control allowed gases, particularly methane, to build up, and disastrous and recurrent explosions in mines literally and figuratively rocked the industry. The horrors of these continuing mine disasters did more than anything else to rouse public opinion and reform the industry in the latter half of the nineteenth century. The successful use of a centrifugal fan in 1849 led the way to improved ventilation, though for many years after that exponents of ventilation, by having two shafts with a furnace at the bottom of the upcast shaft, claimed that this—the hitherto orthodox method of the times—was as good as mechanical ventilation with a fan. One or two customs of early English coalmining are interesting. At Newcastle putrescent fish were taken underground in "gassy" pits so that their phosphorescence would give some light, and in this way use of a naked light underground was avoided. Sometimes an attempt was made to explode small quantities of methane purposely, to prevent its building up into large concentrations. The man who undertook this extremely hazardous work was called a fireman. Muffled in well-saturated clothing he crept along with a light on the end of a long pole, never knowing from moment to moment whether or not the next explosion would be his last.

Socially and economically, times were hard for the coal miner as well as for many others. Hours were long, conditions of work bad and dangerous; many seams were only narrow and the miner worked crouching or lying down; the women and children pulled trucks and carried coal. In some districts "bond" systems of employment and payment in goods from employer-owned shops and inns prevailed. Company-supplied housing was poor on an average, and at its worst little more than a collection of hovels. The miners were often typical products of this environment and were said to be insolent and turbulent and addicted to drinking, fighting and gambling. Accounts of the industrial conditions of the times cannot fail to produce a thrill of horror even at this distance in time. However, it is typical of Victorian England that as the evils of this child and female labour racket were gradually exposed, the subsequent popular outcry arose not so much because of revulsion from the abhorrent material conditions, but because the system prevented the children's receiving religious instruction, and because the fact that nearly naked women and men worked together underground was thought to be conducive to immorality.

Occupational chest disease in English miners prior to the nineteenth century was apparently no great problem, for there are but two medical references to respiratory conditions among them: Percival states that disease sometimes occurs in Derbyshire miners from the inhalation of fine dust, and Beddoes in 1799 thought that millers, stonemasons and certain miners became tuberculous owing to the dust they breathed.

Medically the nineteenth century commenced quietly in the mines. Then in 1822 Patissier, a Frenchman, described epidemic anæmia (hookworm), asthma and rheumatism as diseases of coal miners; Gobert, a Belgian, followed in 1827 by stating that miners' asthma was due to inhaled dust. Finally J. C. Gregory (1831) in Scotland reported "a case of peculiar black infiltration of the whole lungs, resembling melanosis". From henceforth for many years the burning question was whether or not the coal miners' "black spit" and pulmonary melanosis were exogenous or endogenous; that is, did they come from inhaled coal dust, or were they something the lung tissues manufactured? The continental workers were mainly pathologists (Virchow was among them) who were interested chiefly in tissue changes. The Scottish school, on the other hand, were physicians who had to live among their patients and who therefore tended to approach the matter from the "field" angle, and realized only too well that it was an engineering, social and economic problem primarily concerned with ventilation in the pits.

Eventually what commenced as an investigation of a peculiar form of melanosis ended up as a reasonably accurate pathological and clinical concept of silicosis and coal miners' pneumoconiosis. By the time of Brockman (1845) he, together with Thackrah, who had written some years earlier (1832), had recommended most of the things now regarded as necessary but still in some cases not carried out—that is, improved pit ventilation, wetting the dust, preemployment medical examinations, periodical medical examinations, proper bathing facilities, and so on.

Until the mid-point in the century it could be said that serious respiratory disease among all types of miners was high, and that their mean duration of life was less than the average mean duration. Just how bad the problem was cannot be accurately ascertained owing to lack of statistics. In the second half of the century, however, better records became available, and then occurred what to our eyes looking backwards seems to be a strange phenomenon. The "black spit" gradually disappeared, to such an extent that as a medical entity by the end of the century it was almost forgotten. Meiklejohn remarks:

> So it would appear that an end had been reached; the lung diseases of coal miners variously denominated as black spit, black infiltration of the lungs, spurious melanosis, anthracosis, phthisis melanotika and pneumoconiosis anthracosica had, for all practical purposes, ceased to exist as a medical problem. This happy state had been achieved in 50 years 1825 to 1875, by improved ventilation and improved sanitary conditions in coal mines together with shorter working hours.

The public outcry created by numerous pit explosions, and the alarm engendered by the publication of reports of several commissions and inquiries led through the years to a deal of legislation. The first Mines Inspector was appointed in 1843, and the Royal School of Mines was founded in 1851. Both these events had a profound influence on safety and health in future years. I suspect also that successful use of mechanical ventilation in pits did more than all the above to effect improvements.

And so the "black spit" passed into history—so much so that Collis (1915), in a Milroy Lecture, said of coal miners: ". . . fifty years ago asthma was common among them" . . . "the disease, however, common though it used to be, has passed almost unobserved from our midst"; and J. S. Haldane until his death averred in effect that coal miners did not get pneumoconiosis. As recently as when I did my medical training "coal miners did not suffer from any special pulmonary disorder". The only voice crying in the wilderness belonged to Charles Badham, of Sydney, who in the years after the first World War laboured under no illusions on this subject. Actually official statistics in the United Kingdom supported his contention, for, though the disease was no longer a ravage, coal miners all during

this period of fifty years died in excessive numbers from "bronchitis".

At the end of the nineteenth century only 1·5% of the English coal industry—an industry which had made possible the industrialization of England—was itself mechanized. In the first quarter of the twentieth century, however, pits became deeper and seams narrower, and mechanization was introduced without adequate dust suppression. During the last World War the resulting harvest ripened and was garnered. Between 1931 and 1948, 22,000 British coal miners had to leave the pits on account of pneumoconiosis. In the two years ended 1950 an additional 10,000 cases were diagnosed, and 4500 of these men had to leave the industry. In 1945 there were 5706 deaths among coal miners from occupational chest disease. The total work force at this period was about 700,000 men.

Other countries have much the same story to tell. In New South Wales for the five years ended June, 1946 (Nisbet), there were 17,600 miners underground, and on the surface during this period 242 were classed as partially incapacitated and 189 as totally incapacitated. The total incidence of men with incapacitating pneumoconiosis was 4·9 per 1000 colliery employees per year.

In Queensland (1952) we had 3756 men scattered among 95 pits. Many of the seams are damp. Owing to the natural circumstances of shallow and in many cases moist workings, the dust problem is not a severe one. Only one or two mines may give cause for concern. This is not to say, however, that in the years to come catastrophe cannot overtake us. We have history to warn us on this subject. However, for the world at large we can say that in the course of a hundred and fifty years the wheel has made a complete turn and the "black spit" is with us once again.

Siliceous Holocaust.

This intense interest shown in melanosis led as a by-product to an interest in all the health problems of mining communities, including those in Cornwall, and in the latter half of the nineteenth century what was originally a side avenue became the main highway and furnished the chief occupational medical problem for the next one hundred years. I refer to silicosis.

Thackrah (in "The Effects of Arts, Trades, and Professions and of Civic States and Habits of Living, on Health and Longevity", Second Edition, 1832) had pointed out that limestone workers had greater longevity than sandstone workers; but no one quite realized the world of difference between exposure to coal dust and exposure to a dust containing large amounts of free silica. Gradually data concerning Cornish and other metalliferous workers came to light. In the Report of the Royal Commission (1842) evidence was published to the effect that many underground Cornish miners died of galloping consumption at an early age, and that many succumbed to "phthisis" and became incapable of carrying on their occupation between the ages of thirty-five and forty-five years. Of the lead miners of Northumberland and Durham, it was stated that after thirty years nearly all had chest complaints and a poor life expectancy. In this area, of 79 miners who had lived to forty-five years or over, 37 had died of consumption. In certain villages the number of deaths due to chest diseases was about 50% of the total mortality.

After the turn of the half century better statistics became available. In 1858 J. B. Thomson stated that the average duration of life among miners could be roughly set out as follows: coal miners, 26·1 years; labourers (undefined but non-mining), 34 years. He also stated that in Cornwall a fair comparison of deaths among the general population and the men in the mines was as follows: 31% of the general population, 61% of the mining population.

Barham (1871) gave the following figures: Of every 10,000 males who attained the age of fifteen years, there were registered as dying of all lung diseases: (i) for all occupations, in England, 2866; (ii) for miners: in Cornwall, 5596; in Stafford, 2655; in Durham, 1958; in South Wales, 3037.

In 1892 Ogle showed that the health of Cornish miners had not improved. Death rates were given as shown in Table I.

There are also other statistics around this time which showed clearly how dangerous tin mining was to the lungs. Also about this period Eulenberg (1876) had drawn attention to the importance of the quartz content of dusts when their injurious properties were being considered. Henceforth industrial hygienists were for nearly fifty years so occupied with various theories concerning the action of free silica in the lungs that they had not the eyes to see the other injurious dusts around them. Koch's isolation of the tubercle bacillus and its obvious association with silicosis in many patients accentuated this trend.

TABLE I.

Age 25 to 65 Years.	Phthisis.	Lung Diseases.	Total Deaths Due to Lung Diseases.
Cornish miners	348	231	579
Coal miners	64	102	166

In the meanwhile metalliferous mining was developing on a vast scale in many parts of the world: gold in South Africa, California and Australia; copper in South America, Spain and the United States; tin in Bolivia; and so on. We know from our own history the vast differences the "gold rushes" made to Australian social economy. These discoveries also had an effect in reverse on Cornwall. Between 1851 and 1891, 170,000 Cornish miners emigrated overseas, taking with them their own peculiar disregard of all forms of dust suppression. We are all familiar, thanks to the art of Oswald Pryor, with the lighter vein in this vast migration of skilled technicians, who took with them across the globe several centuries of mining tradition. We do not, however, often think of the reverse side of the picture; of the graves of the many who died in the prime of life in places scattered around the world, just as surely and as quickly as they would have died on the Gold Coast or in other celebrated "white man's graves". In their case, however, little fame has carried into history, for their deaths cannot be blamed onto a climate or to disease-carrying insects, but to man's avarice and ignorance.

Treve Holman (1947) makes the following statement:

> As a result of the appalling health conditions under which they were compelled to work overseas many hundreds returned to Cornwall every year to die of "miners' complaint". The pallid face and the hard short cough were a well known sight and sound in the Cornish towns.

With these discoveries—or perhaps I should say spurred by these discoveries—of metalliferous riches, much time was devoted to perfecting rock-drilling machines and other forms of mine mechanization. While silicosis was a serious menace before the extensive use of drilling machines, and while other mine operations produce dust, I have always considered that the biggest single factor in producing the holocaust of silicosis that occurred in the metalliferous mines was "dry drilling"—that is, mechanical drilling without the use of water suppressives. Ingersoll and Rand (1870), Holman (1880) and the "Climax" some years later were more or less the order of appearance of the principal drills. Today these three companies (Ingersoll and Rand having amalgamated) are still the chief manufacturers. For many years no water was used at all; then water was thrown on the drill with a jam tin, and later, about 1900, a water spray independent of the drill was used. Between 1893 and 1896 Leyner, the genius of the rock drill industry, radically altered drill design by adopting a hammer type mechanism. From the health point of view the important change was the blowing of dust out of the hole drilled in the rock by passing air down through a hollow in the drill steel. This created so much dust that even miners fairly inured to and unworried by dust refused to use

his drills. He then hit on the idea of passing both air and water down the hollow in the drill steel. This was immediately successful, and so we passed from "dry drilling" to "wet drilling". For many years he had the patent for this, so that his rivals could not use the idea. In 1913 the patent expired. Modern modifications, however, do not require this patent, for now air is not passed down the steel at all, but water only. The air is vented out through ports on the front head of the machine. Today, of course, dry drilling is prohibited in most situations, mine ventilation has been greatly improved, ore is handled wet, and so on. As well as this, in the leading metalliferous mining countries the working force is under strict medical supervision. Unfortunately, where water is scarce there is still a temptation to "drill dry".

What number of silicotics the period 1850 to 1920 produced will never be known with any certainty. However, we have some fragmentary pointers. Mining on the Witwatersrand on a large scale must have commenced between 1870 and 1880. Silicosis appeared within about ten years, and at the worst period, shortly after the Boer War, the active life of a miner was said to be three to five years. Rock-drilling machines were numerous, and drilling must have been for the most part "dry". In an official report in 1902 it was stated that of 1777 rock drill men employed on the Rand before the Boer War, 225 were known to have died of "phthisis". As late as 1937, 70% of the silicotics found on the Rand were machine men. The establishment of the Miners' Phthisis Medical Bureau of Johannesburg is now a part of history. The liability there to contract silicosis since 1916 has been reduced greatly. It could be argued that this reduction is as much as 90%. There is, however, still a small but significant incidence, which would seem to indicate that one cannot completely eliminate the hazard. Incidentally, the Bureau in 1944 could state that it had clinical and radiographic records of 188,000 European and 58,000 native labourers. Many thousands of records are correlated with necropsy findings.

With the limited facilities available I cannot discover what was happening in this period in the Americas. Australia furnishes some meagre records. In 1911 there was a Royal Commission upon the health conditions in Queensland mines. The report sets out the conditions shown in Table II.

TABLE II.

Deaths of Miners from Phthisis and Other Lung Diseases: Rates per 10,000 Living Miners.

Miners.	Phthisis.	Other Lung Diseases.	Total of All Lung Diseases.
Ipswich coal miners (1906–1910)	—	9·6	9·6
Gympie quartz miners (1906–1910)	9·2	21·4	30·6
Charters Towers quartz miners (1906–1910)	39·3	20·6	59·9
Queensland, all miners (1906–1910)	24·9	17·8	42·7
Bendigo miners (1905–1906)	129·6	62·0	191·6
Western Australian miners:			
(1900–1909)	19·21	34·69	53·9
(1909)	31·06	31·19	62·25
Queensland miners (1909)	23·4	24·1	47·5
Tin miners, Cornwall (1900–1902)	—	—	182·00
Coal miners, United Kingdom (1890–1892)	—	—	58·00
Ironstone miners (1890–1892)	—	—	46·00

The Commission rightly observed that the Queensland quartz miner had a lower death rate from lung diseases than the English coal miner at a time when the latter was supposed to have practically ceased to contract an occupational dust disease of the lungs. The death rate per 10,000 of all males in the population aged twenty years and over in Queensland in 1911 from lung diseases was 24·8. It seems obvious, then, that though the Commission was moderately satisfied about the health of the miners, the fact remains that the miner had at least twice the average male's death rate from respiratory disease. In 1952 the incidence in Queensland for a similar age group of all males was 13·1.

This lends some foundation to the tradition that still exists in this State that miners' phthisis was a very real scourge that sooner or later took at least one member of a mining family.

Other statistics available are as follows:

1. In Western Australia, in 1911, of 1805 miners examined 22% had varying degrees of pulmonary fibrosis, and 1·5% had active tuberculosis. In 1925–1926, of 4023 miners examined 16% had silicosis, 3% had silico-tuberculosis, and 0·3% had tuberculosis only. The annual incidence per 1000 men from 1926 to 1949 (22,500 men examined) was as follows: silicosis, 2·37; silico-tuberculosis, 1·0; tuberculosis, 0·36.

2. In Broken Hill, in 1920, of 3917 miners examined 5% had pulmonary disease. Between 1939 and 1949 the incidence has been 0·27% with pulmonary disease.

3. In Queensland admissions to workers' compensation for all forms of pneumoconioses and occupational tuberculosis among miners are as shown in Table III.

TABLE III.

Period.	Number.	Period.	Number.	Period.	Number.
1917–1918	26	1929–1930	129	1941–1942	44
1918–1919	98	1930–1931	89	1942–1943	35
1919–1920	47	1931–1932	99	1943–1944	46
1920–1921	51	1932–1933	50	1944–1945	50
1921–1922	238[1]	1933–1934	39	1945–1946	39
1922–1923	124	1934–1935	39	1946–1947	47
1923–1924	94	1935–1936	41	1947–1948	65
1924–1925	125	1936–1937	30	1948–1949	65 [2]
1925–1926	169	1937–1938	42	—	
1926–1927	167	1938–1939	51	1949–1950	38
1927–1928	166	1939–1940	52	1950–1951	31
1928–1929	122	1940–1941	50	1951–1952	27

[1] Prior to July 17, the Treasury was paying for miners' phthisis 10s. a week, called a "compassionate grant"; 183 such cases were admitted in 1921–1922, when the Act was widened.

[2] Included readmissions due to a change in the Act.

Conclusion.

There are approximately 7000 miners — coal and metalliferous — in Queensland, including men working above ground. The incidence of annual incapacity per thousand miners per year is now approximately four. From personal experience I know, however, that this is not a correct indication of what is happening at present. Many of the claimants awarded compensation are old metalliferous miners, well advanced in age, who have long ceased to have any connexion with mines.

I think, however, we can at least state as far as South Africa and Australia are concerned that an end was reached between 1920 and 1930. The metalliferous mining industry has been steadily putting its house in order. As producers of gold the glories of Victoria and Queensland are departed; in Western Australia and at Broken Hill engineering and medical control has reduced the incidence to small proportions. There is still a definite incidence in secondary industries among sand blasters, foundry men and the like; but the numbers exposed compared to the numbers engaged in mining are not so great, and in the last ten years most secondary industries have either voluntarily or compulsorily reduced their hazard.

My sketchy tale is now complete. Many will be indignant that I have not mentioned the dust hazards in secondary industry. They are varied, interesting and usually amenable to control. Time and space, the masters of us all, have made this omission mandatory. I also think that mining has provided and nearly always will provide the main single environment in which man contracts his worst occupational disease.

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SUICIDES IN TASMANIA.

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SINCE I read in 1941 an article by E. H. Derrick in this journal on "Suicide and its Prevention", this subject has continued to interest me—particularly as Tasmania has the lowest suicide rate of all Australian States.

The subject of suicide presents many facets, and an attempt was made to elucidate some of them. The only materials available were the depositions made at coronial inquiries. Sometimes these failed to reveal an important fact such as the age of the individual, but generally the relevant information was readily extracted. Only those cases in which the coroner returned a finding of suicide were considered, and no doubt some in which an open verdict was given were, in fact, cases of suicide. This limitation tends to load the figures a little; for example, a coroner might hesitate to find suicide in a case of drowning unless there was other evidence to support this finding; but what coroner would hesitate when presented with the usual facts attending a "cut throat"?

A twenty-year period was selected, from 1934 to 1953 inclusive. During this time there were just over 400 cases of suicide in Tasmania; but as some inquest depositions were not available for study the number examined for this paper was 382. These suicides took place in various parts of the State. In nearly all cases an autopsy was performed, in many of them by myself.

The figures obtained are given below, the presentation being similar to that in Derrick's article, so that a comparison with Queensland can readily be made.

The age and sex incidence follows the usual pattern, with males predominating. There were 289 males to 93 females, giving a ratio of 3·1 to 1·0. This predominance is evident at all ages. The ages of the suicides varied from a boy aged twelve years to an old man aged eighty-seven. The age distribution was similar to that in Queensland, but twice as high in the youngest group. Like cancer, suicides increase with age.

The monthly and seasonal totals of Tasmanian suicides over twenty years are shown in Table II. As in Queensland, the rate is lowest in winter. This is interesting, because it was suggested that in Queensland the decrease in winter was due to the increase in social activities rather than to the lowered temperature. Were this so, one would have expected an increase in Tasmania during our fairly severe winters when social activities are relatively restricted. I am quite at a loss to explain why our rate in the spring is one and a half times that in the winter.

In some cases the time of death was unknown, but in others it could be given exactly:

A sixteen year old boy left the following note: ". . . This is the first and last shot I shall have out of Dad's rifle. The time of this happy ending is 10.10 P.M."

The driver of a train was horrified to see a man crawl forward and place his head on the railway line. The time was exactly 10.38 p.m.

However, when possible, the time of the suicide was noted, and a distribution was made over eight periods of three hours each. In spite of the fairly frequent association of suicide and insomnia, the frequency in the period 2 a.m. to 5 a.m. was easily the lowest (Table III).

The methods employed to end life are set out in Table IV. They are similar to those in many other published series.

Firearms comfortably head the list, and this is to be expected in a country where no licence is required to own a rifle or shotgun, and where shooting is a popular sport.

TABLE I.

Suicides in Tasmania: Age and Sex Incidence.[1]

Age Group. (Years.)	Males: Number of Suicides.	Males: Number of Suicides Divided by Percentage of Population in Age Group.	Females: Number of Suicides.	Females: Number of Suicides Divided by Percentage of Population in Age Group.
10 to 19 ..	13	0·80	5	0·31
20 to 29 ..	40	2·55	7	0·45
30 to 39 ..	44	2·93	24	1·63
40 to 49 ..	51	4·32	17	1·52
50 to 59 ..	44	4·90	21	2·16
60 to 69 ..	41	6·12	14	2·01
70 to 79 ..	25	8·64	2	0·61
80 and over ..	13		1	
Age not stated ..	18	—	2	—
Total ..	289	—	93	—

[1] The average suicide rate (over a nineteen-year period) per 100,000 of population is as follows: males 11·74 and females 4·03; this gives a combined rate for both sexes of 7·94.

It is interesting to note that females comprise over 10% of those who committed suicide by shooting—relatively a very high figure. Bullet wounds were inflicted by ·22 calibre rifles, with an odd pistol, revolver or service rifle. Of the 66 bullet wounds, 57 were in the head and nine in the chest. Of 50 bullet wounds in the head, 22 were directed from the side, 19 from the front, seven through the mouth, one through the nose, and one entered the skull from under the chin. In no case was the eye pierced; such an injury therefore would suggest murder or accident rather than suicide. Shotguns were not used quite so commonly as rifles. In the 47 cases, the gun was directed to the head 27 times, to the chest 13 times, to the neck six times, and to the abdomen once. Suicidal shotgun wounds, being received from close range, cause tremendous destruction, particularly to the head.

Falls from a height were quite rare—no doubt because the Hobart Bridge is a floating one.

TABLE II.

382 Suicides in Tasmania—Monthly and Seasonal Incidence.

Period.		Number of Suicides.	
Winter:			
June	.. .. ..	30	78
July	.. .. ..	24	
August	.. ..	24	
Spring:			
September	.. ..	32	121
October	.. ..	40	
November	.. ..	49	
Summer:			
December	.. ..	37	85
January	.. ..	27	
February	.. ..	21	
Autumn:			
March	.. .. ..	40	98
April	.. .. ..	31	
May	.. .. ..	27	

Cut throats were fairly common in men, but rare in women. Is it that women do not like knives, or is it that a woman is naturally averse to making a mess?

In spite of easily accessible water-ways in most areas, drownings were relatively few; perhaps the water is *uninviting here.*

TABLE III.

288 Suicides in Tasmania: Time of Act.

Time.	Number.	
2 a.m. to 5 a.m.	13	
5 a.m. to 8 a.m.	34	145
8 a.m. to 11 a.m.	55	
11 a.m. to 2 p.m.	43	
2 p.m. to 5 p.m.	40	
5 p.m. to 8 p.m.	36	143
8 p.m. to 11 p.m.	28	
11 p.m. to 2 a.m.	39	

Hanging is a popular and very effective method, and was rather more commonly used by females than might have been expected. *In a series of 69 hangings, the following* were used: rope 57 times, belt or strap six times, wire twice, electric light cord once, and clothing or bedding three times. In a series of 58 hangings, an outhouse was chosen 35 times, the house 13 times, and some outdoor structure such as a tree 10 times.

Poisoning was used less often than in many other series, but, as expected, was the method most commonly used by females (Table V).

Of the 16 suicides by carbon-monoxide poisoning, in seven a gas oven was used *(sometimes with a pillow)*, and in six, exhaust gases from a motor-car.

TABLE IV.

382 Suicides: Method Employed.

Method.	Males.	Females.	Both Sexes.
Firearms	100	13	113
Explosives	1	—	1
Drowning	36	24	60
Hanging	65	16	81
Fall from a height	4	4	8
Cut throat	28	2	30
Run over by train	1	—	1
Cut vessel	3	1	4
Strangulation by rope	1	—	1
Throwing self under car	1	—	1
Knife wound to chest	1	—	1
Poisons	48	33	81
Total	289	93	382

Fourteen persons used a double method. (Only the principal method in each case is included in Table IV).

Four men cut both wrists and throat. Two others cut their throats, and then hanged themselves—and one of these cut his wrists as well.

Another man cut his axilla and then his throat.

A carpenter, aged forty-eight years, first cut his wrists, and then took rat poison. A commercial traveller, aged twenty-eight years, cut his wrists and then broke open a gas meter.

A woman, aged fifty-three years, took gentian violet before drowning herself. Her mouth showed violet-coloured froth.

A returned soldier, aged forty-two years, took "Lysol", and then put his head in a gas oven.

A man stood on the end of a diving board, and shot himself through the head, using the same rifle as his wife had used to commit suicide some time before. His body was found in the water. There was fluid in the lungs.

A widower, aged fifty-six years, after assaulting a girl, shot himself in the chest and head with a ·45 calibre pistol.

A poultry farmer was found with a ·22 calibre bullet wound *in his forehead, and a 12 gauge shotgun wound in the left* side of his chest. Murder was suspected, but investigation showed it to be a clear and determined case of suicide. The *bullet entered the base of the skull, but the brain* was undamaged.

Threats and Previous Attempts.

In this series there was a record of a threat to commit *suicide in 115 cases, and a previous attempt* had been made in 33 cases. No doubt the true figure for both would be considerably higher. Sometimes the threat was vague and not taken seriously; *but sometimes it was definite and* often repeated.

A man, aged sixty-eight years, who ultimately fell from a window, had threatened suicide by poisoning, jumping, electrocution, and cutting. Poison and razor blades were found *concealed in his garden.*

Occasionally the threat was taken seriously, but little effort was made to prevent the act.

A miner, aged forty-four years, left a note, then quietly shook hands with his brother and his wife before taking a *fatal dose of "Lysol". He had previously* threatened and attempted suicide.

TABLE V.

81 Cases of Suidical Poisoning.

Poison Used.	Males.	Females.	Both Sexes.
"Lysol" (and similar compounds)	19	14	33
Carbon monoxide	10	6	16
Strychnine	7	4	*11*
Arsenic	1	2	3
Barbiturates	3	2	5
Cyanide	1	—	1
Phosphorus	2	1	3
Hydrochloric acid	1	—	1
Morphine	1	—	1
Chloroform	—	1	1
Nicotine	1	2	3
Potassium permanganate	—	1	1
Camphor	1	—	1
Hexone	1	—	1
Total	48	33	81

Notes.

Suicide notes were found on 86 occasions, and proved to be of great interest. They simplify the duties of a coroner, often give an insight into the mental condition of the victim at the time, and may indicate the reasons for the act. Notes were written on a variety of materials and usually in pencil. They varied in length from several pages down to a single word. Some notes were more or less indecipherable, while others were lucid and precise. One woman *left a long note* with details of her possessions and their distribution, neatly arranged her husband's clothing, and even left him a reminder to put out the rubbish bin. One of the most interesting notes was written by a man who took strychnine. As the poison began to take effect, he added to the note, and the jerkiness due to tetanic spasms became painfully obvious.

Some typical extracts from suicide notes are as follows:

> Goodbye dear children, please forgive me.
> . . . I have tried and failed.

> Forgive me for this. I cannot carry on. I will be found at . . .

> Life's no good.

> I cannot live any longer. God help my dear wife and relatives.

> . . . Love to all. Will you bury me at . . .

On at least two occasions notes were left warning others of the danger of entering because of the fumes. Some notes had a "legal flavour", as in a letter addressed to the coroner:

> This is to say that this day I have committed suicide whlie of perfectly sound mind through financial trouble.

Considerable ingenuity was shown by the man who loaded a cartridge into the right barrel of a shotgun, and a suicide note into the left. He was careful, too, to pull the appropriate trigger.

Causation and Motivation.

An attempt was made to arrive at the main underlying cause of the suicide in each case. While this was obvious in some cases, in others the act was wholly inexplicable. In 114 cases, the cause was not sufficiently obvious to be given a name. The possible ætiological factors are summarized in Table VI.

TABLE VI.

382 Suicides: Possible Ætiological Factors.

Possible Ætiological Factors.	Number of Suicides.
Nervous disease (some subjects quite insane)	97
Impaired health	78
Marital, domestic and sex problems	37
Financial and business worries	29
Crime	9
Cancerophobia	7
Senile changes	7
Suicide associated with pregnancy	4
Unknown	114

It will be noticed that Table VI does not include alcoholism as a cause. There was certainly a close relationship between alcoholism and suicide, for in no less than 65 cases the deceased was a chronic alcoholic, or definitely under the influence of alcohol at the time. (When the 65 cases were divided into two ten-year periods, it was found that 17 occurred in the first decade, and 48 in the second—that is, the post-war years.) However, it was considered that alcoholism should be regarded in most cases as a symptom of the underlying disease rather than as the cause of the suicide.

After reading through the thousands of pages of depositions, and seeing these people in the mortuary, one cannot help being impressed by the misery and the conflicts which the deceased must have experienced before the mental balance was finally overthrown. Yet in many cases the conflict was only of a minor nature, and should never have been allowed to assume the importance that it did.

A schoolboy, twelve years old, was reprimanded by his mother for a minor offence. Not long after, the son asked his mother to forgive him and to be friends again. She declined. Five minutes later she found him hanging in the wash-house.

The post-mistress in a small country post-office, poorly paid and in indifferent health, was found to be £50 short in her books. Rather than face the problem she ended her life with poison.

Although the subject of a suicide is not uncommonly used in jokes—as in the cartoon of the very sedate butler addressing himself to his master hanging by the bell rope, "You rang, Sir?"—few instances of humour were found in this series, and each was an example of unconscious humour.

At an inquest, a de-facto wife described how she and her baby had been beaten and ill treated by the deceased "husband". She then went on to say: "I entered the shed and saw him hanging. I did not cut him down."

One November a man left a suicide note on the back of a fortune-telling card. The front of the card read: "November should be your lucky month . . ."

One man may have peeped into the future when he left this note: "Open me up and see what I have gone through. They say I look well, but I have put in years of silent suffering."

The shortest suicide note carried only one word: "Goodbuy."

Discussion.

This paper leaves unanswered several important questions, such as the following:

1. Can suicides be prevented? I agree with Derrick that the establishment of psychiatric clinics could lower the suicide rate. Such clinics are attempting to face this problem, especially in Launceston and Hobart. There is certainly wide scope for such clinics, for the high-pressure living of today soon finds out those who cannot adjust themselves to the strains. Many of these will fall behind, and some will suicide unless they can be helped.

2. Is suicide less common among Jews and Roman Catholics than among Protestants, as has been stated? As the religion of the unfortunate people in this series was not disclosed, no answer can be given.

3. Does the reporting in the newspapers of the details of suicides increase the suicide rate? I cannot believe that it does, although there seemed to be a tendency in this series for a particular mode of suicide to recur in cycles. Reporting details of suicides could, in my opinion, increase the rate only by indicating to would-be suicides those methods which are proving most successful.

Summary.

Three hundred and eighty-two suicides in Tasmania occurring over a twenty-year period are analysed. The various methods employed, the frequencies and the time of the day and year are tabulated, and an attempt is made to indicate some of the ætiological factors.

Acknowledgements.

I am indebted to Dr. R. J. D. Turnbull, Minister of Health for Tasmania, and to Mr. R. Fagan, Attorney-General, for permission to publish this paper; to the Government Statistician for some population figures; and to Miss E. Todd, Miss D. Kurth, Miss N. Eadie and Mr. B. Murphy for their assistance in extracting the depositions.

ENDOMETRIOSIS AND STERILITY.[1]

By ROBERT MACKEY,

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THE very nature of the pathogenesis and sequelæ of endometriosis, both internal and external, should decrease considerably the chances for successful occurrence and maintenance of pregnancy. Cyst formation in and around the ovaries, interference with follicular rupture, hæmorrhage, fibrosis and adhesions, in addition to the natural avoidance of intercourse due to the almost constant dyspareunia, are factors which must produce a sterility pattern. The normal fertility rate in marriage is estimated at 88%, but the relative sterility rate has been estimated at 48·9% in patients with endometriosis and the absolute rate at 32·1% (Counseller). The role of internal endometriosis is not discussed in this paper, which deals solely with the external variety. The patients are those who have come through the Sterility Clinic of The Women's Hospital and the private practices of Dr. Alan M. Grant and the author; all have been proven to have endometriosis at operation and have had a complete sterility survey.

Incidence.

Three thousand consecutive sterility clinic cases were analysed. In 18 endometriosis was proven to be present at operation—a known and absolute percentage incidence of 0·6.

However, in 32 other cases there were symptoms and signs which led the examining physician to suspect and record endometriosis, generally on the symptom pattern. If these two groups are combined, the possible incidence in this series is 1·6%. When compared with that in a group of gynæcological patients the incidence is low.

[1] Read at a meeting of the Clinical Society of the Women's Hospital, Crown Street, Sydney, on December 3, 1953.

Scott and Te Linde (1950) report a 9% incidence with an absolute sterility rate of 33·5%. Most investigators agree that the number of cases of endometriosis is increasing, particularly amongst private patients, and the sterility clinic patient approximates closely to these. There appears to be no reasonable explanation of this increase in rate.

Age of Patients.

The average age in the clinic group was 28·1 years, and in the private group 32·2 years; the oldest patient was aged thirty-seven years and the youngest twenty years. In the public group only four of the 18 patients were thirty years of age or more; 17 of the 25 private patients were in this age group. As the period of sterility in both groups showed little variation, the greater incidence may be due to the tendency of the higher income groups, from which I assume the private patients came, to marry and reproduce later. The average age of the clinic patients was 27·6 years for the 3000 under consideration.

Duration of Sterility.

The average duration of sterility was 2·5 years, the longest period being fifteen years and the shortest one year.

Actually the private patients had a lower than average period of sterility before seeking advice, namely 1·9 years, whereas the average for the clinic patients was 3·4 years; this latter figure is high on account of one patient with a history of fifteen years' sterility.

Previous Pregnancies.

Of the 43 patients, 37 had never been pregnant. Two of the patients had had two children previously; one had had one child; three had had two miscarriages each. If a fertility rate could be determined pre-operatively it would be 14%. Haas (1951) assessed a figure of 8·6% for his patients.

Previous Operations.

Thirty patients had undergone no previous abdominal surgery. One patient had had a cholecystectomy, seven appendicectomy, and the remaining six sections for pelvic surgery, three of these for endometriosis and the remaining three for unknown conditions; of the three who had had endometriosis, the disease had recurred in two.

Symptoms and Signs.

Naturally, the main reason for seeking advice was sterility; but symptoms and signs of endometriosis could be elicited in a number of cases. Eighteen patients gave a history of dyspareunia, dysmenorrhœa or menstrual irregularities, whilst in 36 cases there was clinical evidence, such as a fixed retroversion, tender fixed ovary or ovarian cyst, or thickening and nodules in the utero-sacral ligaments or recto-vaginal septum.

Tubal Patency Tests.

Rubin's test is recorded as having been performed on 37 patients; 26 gave a normal tracing, in seven there was evidence of stenosis, and in four the Fallopian tubes were not patent. In conformity with a policy in which salpingography is performed despite a positive Rubin's test result (*a*) in the presence of gross pelvic disease as ovarian cysts or (*b*) when pregnancy has not occurred within three months of the original Rubin's test, hystero-salpingography was performed in 31 cases. This revealed tubal damage in 15 cases, mainly at the fimbriated end of one or both Fallopian tubes. With Rubin's test 30% of the patients showed tubal abnormality; with salpingography the figure was 48·4%.

Treatment.

In all cases conservative surgery was performed, cysts being removed or resected and areas of endometriosis excised, and in all cases the uterus was ventro-suspended. In the whole series there was no need for any major castrating operation. Hormonal treatment was not attempted.

Pregnancies.

Of the 45 patients, 21 became pregnant, an incidence of 46·6%; and as a number of the operations have been performed recently, a further improvement in this figure can be anticipated. Amongst the clinic patients there were eight pregnancies, compared with the over-all pregnancy incidence in the clinic of 981 pregnancies in 3000 investigated cases. There were nine pregnancies amongst the 32 patients with symptoms and signs of endometriosis, which was not, however, confirmed by operation. If these are true cases of endometriosis, which is doubtful, then operation would give a somewhat better chance for a pregnancy. The latent period from operation to pregnancy where this is recorded (in 13 cases) was ten and a half months. Pregnancy was much more likely to occur when there had been no previous operations (13 cases) or simple appendicectomy (six cases). In two of the pregnancies miscarriage occurred, and one patient had a still-born baby at term. There were no ectopic pregnancies.

Discussion.

It seems from a consideration of sterility patients with symptoms and signs of endometriosis that operation produces their greatest chance for pregnancy. The diagnosis is generally available from a consideration of the clinical history, symptoms and signs. Evidence of tubal damage provided by Rubin's test or salpingography may be confirmatory, although normal test results do not exclude it. It is urged that pelvic tuberculosis be excluded by endometrial biopsy and culture prior to surgery being undertaken, as the clinical symptoms and signs of the two diseases may be closely allied. The use of the culdoscope will aid in the diagnosis, although technically the well-advanced case of endometriosis presents an almost insuperable barrier to this method of investigation.

Summary.

1. A review of 45 proven cases of endometriosis in sterility patients is presented.

2. In all cases the patients were submitted to conservative surgery.

3. In 21 of the 45 cases the patients became pregnant.[1]

Acknowledgements.

The author's thanks are tendered to Dr. Alan M. Grant for his help and for access to his private records, and also to the staff of the Sterility Clinic, The Women's Hospital, Crown Street, Sydney.

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RESECTION OF LUNG TISSUE IN THE TREATMENT OF PULMONARY TUBERCULOSIS.

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THE number of patients undergoing surgical treatment for the control of pulmonary lesions in tuberculosis has greatly increased in the last few years. This is due to a much wider use of resection of lung tissue made possible largely by the increasing availability and use of effective antibacterial substances.

It is proposed to discuss the experiences at the Austin Hospital, Sanatorium Section, with the first 200 patients who had lung tissue resected. The first 100 resections were

[1] In the interval between presentation of the paper and publication two more patients in the series have become pregnant, bringing the total to 23.

completed by November, 1951, and these patients have been followed up for at least eighteen months.[1] The results are reported in detail and compared with recent overseas reports.

The writer has been senior medical officer of the Sanatorium Section since 1946 and has himself conducted the follow-up clinic. The thoracic surgical team has grown from one surgeon before 1947 to four surgeons in 1953, each surgeon has operated on a half-day session per week, and all attended together with physicians from local sanatoria at a weekly review for final selection of patients for surgical treatment.

Table I shows the numbers of patients undergoing surgical procedures in the years 1947 to 1953. It will be seen that in 1947 22 patients were treated surgically and in 1953 the number was 157. The table also shows that resection has been used increasingly as an alternative to thoracoplasty and other forms of major collapse therapy.

TABLE I.

Major Thoracic Surgery, Years 1947 to 1953.

Year.	Resection.	Thoracoplasty and Its Modifications.
1947	0	22
1948	10	34
1949	17	43
1950	29	53
1951	51	56
1952	80	40
1953	120	37

Indications for Resection.

The following is a list of the indications for resection that it is thought are generally accepted, provided that remaining lung tissue is free from disease, or that any disease present is completely quiescent: (i) large tuberculoma, (ii) "blacked out" destroyed lung, (iii) bronchostenosis with infection distal to stenosis, (iv) "resistant" lower lobe cavities, (v) "failed thoracoplasty".

Patients suffering from the first three conditions would not be benefited by thoracoplasty; "resistant" lower lobe cavities have in the past, both in this unit and in other centres, been treated by surgical collapse, but most surgeons now prefer resection. After "failed thoracoplasty", resection is preferred to revision of the thoracoplasty.

Whether resection should be performed in preference to thoracoplasty for apical disease when either form of treatment could be used is still a matter for argument. The majority of American centres seem to prefer primary resection in such cases.

In England the position is still undecided. Holmes Sellors in 1952 stated that he still preferred thoracoplasty to resection when thoracoplasty appeared to offer a reasonable chance of success, but Bickford *et alii* (1952), with an experience of 729 resections, prefer resection to thoracoplasty except when there is extensive infiltration on the opposite side.

At the Austin Hospital resection is used in preference to thoracoplasty, except when there has been widespread involvement of one or both lungs and the patient has been left with a retracting or cavitated apical zone and scattered areas of probably quiescent tuberculosis in other parts of the lung fields.

There is considerable difference of opinion as to the best method of treatment for patients who have persistent localized caseous nodules after intensive antibacterial treatment. In the past many such patients were discharged from sanatoria and a great number of them have remained well. Figures for relapse rates vary in different reports. Ryan, Medlar and Welles (1952) advocate multiple and if necessary bilateral resections to remove all these caseous residues. Our surgeons agree with this in principle, and although they have performed very few bilateral resections they are prepared to do this if necessary. It is thought that any caseous nodule over two centimetres in diameter is dangerous, and a patient with such a nodule has a better chance of remaining well if the nodule is resected.

Occasionally, when all else has failed, the surgeons have resected a small area of disease that seemed exceptionally dangerous and knowingly left scattered disease in other parts of the lungs. For instance, a patient with a persistent cavity in the apical segment of a lower lobe and widespread disease may remain in jeopardy with little or no improvement until the cavity-bearing segment is removed; then further antibacterial treatment may be very effective.

Until recently it was customary to delay major surgical procedures until prolonged sanatorium treatment with antibacterial substances and minor collapse measures had been tried; but now, when it seems likely that localized destructive areas of disease will resist such measures, operation is undertaken early with considerable saving of time for the patient. No resection is undertaken until the patient has had at least a month to six weeks' full antibacterial treatment and radiological observation.

The First 200 Resections.

Table II shows the types of resections which were performed on the first 200 patients. In the first series there were 76 pneumonectomies and 27 lesser resections, and in the second series 24 pneumonectomies and 76 lesser resections. These figures demonstrate the broadening indications for resection which were discussed above.

TABLE II.

Extent of Resection in First Two Hundred Patients.

Procedure.	First Hundred Patients.	Second Hundred Patients.
Pneumonectomy ..	76	24
Lobectomy	23	47
Segmental resection ..	4	29

In the first series three patients had two operations. In each patient spread of disease to the remaining ipsilateral lung tissue occurred after lobectomy and further resection was performed to complete the pneumonectomy. One patient included in the pneumonectomy group had undergone a previous right upper lobectomy at another hospital, and late spread of disease to the remaining lobes necessitated completion of the pneumonectomy.

Detailed Analysis of First 100 Cases.

The indications for operation in the first 100 cases are shown in Table III.

The age and sex distribution are shown in Table IV.

Eighty-five of the 100 patients were alive at the time of review in June, 1953, and Table V shows their state at that time.

The average duration of the follow-up was thirty-five months, with a minimum of eighteen months and a maximum of five years. Sixty-six patients were well and working at that time, and eight more had excellent prospects. Of these 74 patients, 60 had "positive" sputum on direct smear examination just prior to operation, and 30 of them had very little prospects of ever leaving the sanatorium without surgical help.

Complications and Deaths.

Fifteen patients have died since operation; the average period between operation and death was 3·3 months, varying from 0 to 15 months.

The causes of death are reviewed in Table VI.

The total number of deaths in the 200 patients was 16, there being only one death in the second 100, the mortality rate in the whole review being 8%.

[1] Survey of living patients in June, 1953.

TABLE III.
Indication for Resection.[1]

Procedure.	"Blacked-out" Lung.	Extensive Unilateral Disease.	"Failed Thoraco-plasty."	Broncho-stenosis.	Extensive Lobar Disease.	Tuberculoma.	Lower Lobe Cavitation.	Total.
Pneumonectomy ..	22 (7)	38 (4)	9 (1)	7 (1)	—	—	—	76 (13)
Upper lobectomy ..	—	—	1	—	15 (2)	—	—	16 (2)
Lower lobectomy ..	—	—	—	—	1	—	6	7
Segmental resection ..	—	—	—	—	—	4	—	4
Total	22	38	10	7	16	4	6	103 (15)

[1] Number of patients now dead shown in parentheses.

The patients were assessed as good and bad "operative risks" prior to operation. One hundred and sixty-nine patients were good-risk patients and 35 bad-risk patients; this classification has erred on the side of placing doubtful cases in the good risk group.

Three patients in the good-risk group and thirteen in the bad-risk group have died from all causes since operation, the mortality rates being 1·8% in the good-risk group and 37% in the bad-risk group.

TABLE IV.
Age and Sex Distribution.[1]

Age Group. (Years.)	Males.	Females.	Total.
0 to 9	0	1 (1)	1 (1)
10 to 19	2	6 (1)	8 (1)
20 to 29	7[2]	34[2] (3)	41 (3)
30 to 39	7 (3)	21[2] (3)	28 (6)
40 to 49	5	9 (1)	14 (1)
50 to 59	3 (2)	5 (1)	8 (3)
Total	24 (5)	76 (10)	100 (15)

[1] Number of patients now dead shown in parentheses.
[2] One patient in each group had two resections.

Apart from the 15 fatal complications in the first 100 cases, 12 patients had post-operative complications which were controlled. Those patients who recovered are reviewed in Table VII; one patient survived an empyema and a late spread to the remaining lung, and is now well and working.

TABLE V.
Patients Alive at Time of Survey.

Condition.	Number of Patients.
Well, and working, with "negative" or no sputum ..	66
Not working, with "negative" sputum or no sputum:	
(a) Fit for work soon	6
(b) Semi-invalid	7
Home, "positive" sputum:	
(a) Well	1
(b) Semi-invalid	1
Still in sanatorium:	
(a) Well—fit for discharge soon	2
(b) "Positive" sputum and/or present active disease	2
Total	85

Early Spread.—Fresh activity within three months of operation has been classified as early spread attributed to the operation. This occurred in two patients; one who had had a pneumonectomy died. In the other patient extensive contralateral disease followed a lower lobectomy; this disease responded to antibacterial treatment, and a residual apical cavity was closed by thoracoplasty. The patient is now well and working.

Late Spread.—Spread of tuberculosis occurring more than three months after operation has been classified as late spread. It occurred in four patients after pneumonectomy; two are still under active sanatorium treatment with antibacterial therapy, and the other two responded to such treatment and are now well and working. In three patients late spread occurred in the residual ipsilateral lung tissue after lobectomy; each required further resection and is now well and working.

TABLE VI.
Causes of Death.

Cause of Death.	Pneumon-ectomy.	Lobectomy.
Hæmorrhage	2	0
Anæsthetic mishap	1	0
Early spread	1	0
Non-specific broncho-pneumonia with anoxia ..	1	0
Pulmonary embolus	1	1
Fistula and empyema ..	4	1
Congestive cardiac failure ..	2	0
Amyloid disease following subscapular abscess ..	1	0
Total	13	2

Fistula and Empyema.—Fistula and empyema occurred five times in the 103 resections. All five patients were recognized as presenting desperate risks, and fistula was associated with spread of disease which caused the death of the patient. These patients will be discussed in more detail below.

TABLE VII.
Non-Fatal Complications.

Complication.	Pneumon-ectomy.	Lobectomy.
Early spread	0	1
Late spread	4[1]	3
Empyema	4[1]	0
Subscapular abscess	1	0

[1] One of each occurred in the same patient.

Empyema without Fistula.—In four patients empyema occurred without fistula. In two of them empyema followed thoracoplasty to obliterate space after pneumonectomy, and on each occasion the thoracoplasty was performed six weeks after resection, the wound failed to heal completely, and a sinugram revealed a pleural pocket. Both these patients have small sinuses which discharge intermittently with a minimum of discomfort. One patient developed a small localized empyema after pneumonectomy with immediate thoracoplasty; this was drained and the sinus healed in a few weeks and has remained healed. This empyema was a small abscess surrounding the bronchial

stump. The fourth patient developed an empyema following lower and middle lobectomy to complete pneumonectomy, the upper lobe having been resected earlier. As in the first two cases, the wound broke down after operation and has continued to discharge, although the patient is now well and working, with a minimum of inconvenience.

Subscapular Abscess.—Two patients developed subscapular abscess. In one case drainage of the space effected a cure; but in the other, in spite of adequate drainage and repeated exploration for fistula, necrotic rib or any other reason for failure to heal, the patient finally developed amyloid disease and died.

Hæmorrhage.—On two occasions after pneumonectomy on very poor risk patients operative hæmorrhage was excessive and could not be controlled. The hæmorrhage consisted of continuous oozing from all raw surfaces, and in spite of massive transfusion both patients died, one on the operating table and one shortly after leaving the table.

TABLE VIII.
Complications Following Pneumonectomy.[1]

Complication.	Previous Thoracoplasty.	No Thoracoplasty.	Immediate Thoracoplasty.
Hæmorrhage	0	2 (2)	0
Anæsthetic mishap	0	1 (1)	0
Early spread	0	1 (1)	0
Late spread	0	1	3
Non-specific bronchopneumonia	0	0	1 (1)
Pulmonary embolus	0	0	1 (1)
Fistula and empyema	1 (1)	1 (1)	2 (2)
Empyema	1	2[2]	1
Congestive cardiac failure	0	0	2 (2)
Subscapular abscess	0	0	1
Amyloid disease and subscapular abscess	0	0	1 (1)
Number of cases complicated	2 (1)	6 (5)	12 (7)
Total number of patients	11 (1)	25 (5)	40 (7)

[1] Number of patients now dead shown in parentheses.
[2] One patient also had late spread.

On each occasion pre-operative blood examination had revealed no bleeding tendency and the pre-operative hæmoglobin value was above 12 grammes per 100 cubic centimetres. No thoracoplasty was performed on either patient, and it is interesting to speculate whether, if immediate collapse measures had been carried out and the walls of the space allowed to come together, the hæmorrhage might have stopped.

Anæsthetic Mishap.—One patient died as the result of an anæsthetic misadventure. The patient was anoxic for ten minutes, did not regain consciousness, and died sixteen hours after operation.

Non-specific Bronchopneumonia.—Non-specific bronchopneumonia occurred in one patient after pneumonectomy and immediate thoracoplasty, and the patient died of anoxia on the third post-operative day.

Pulmonary Embolus.—Two fatal pulmonary emboli occurred, both in middle-aged patients. No non-fatal embolus occurred in this series.

Congestive Cardiac Failure.—Congestive cardiac failure occurred in two middle-aged patients after pneumonectomy; both died three months after operation. In one case œdema could not be controlled, and during the post-mortem review it was realized that the patient had been having 12 grammes of sodium PAS daily since operation. In the second case cardiac failure was precipitated by *status asthmaticus.*

Variations in Operative Technique.

Operative techniques have changed with increased experience and a better understanding of the problems involved. Our surgeons believe that the space previously occupied by resected lung tissue should be obliterated as soon as possible to avoid over-expansion of remaining lung tissue and to remove a potential empyema cavity. Bronchial closure should be meticulous, the stump should be buried, and no blind bronchial cul-de-sac allowed to remain—again to minimize fistula formation with subsequent empyema.

The importance of bronchial closure has been recognized throughout the series, and all surgeons have paid particular attention to this matter. The most satisfactory method of obliterating residual space is still causing some anxiety after certain types of resection.

Pneumonectomy.—After pneumonectomy a seven-rib thoracoplasty with phrenic nerve interruption and drainage of the pleural cavity is now almost invariably performed at the time of pneumonectomy, and in many cases it is performed as soon as the lung has been freed from the chest wall; this increases exposure and gives time for oozing to cease. In the first cases in this series the thoracoplasty was performed four to six weeks after resection, which was done without drainage.

TABLE IX.
Complications Following Lobectomy.[1]

Complication.	Lower Lobectomy.	Upper Lobectomy: Immediate Thoracoplasty.	Upper Lobectomy with Previous Thoracoplasty.	Upper Lobectomy.
Early spread	1	0	0	0
Late spread	2	1	0	0
Fistula and empyema	0	0	1 (1)	0
Pulmonary embolus	0	0	0	1 (1)
Residual space	0	0	0	2
Number of cases complicated	3	1	1 (1)	3 (1)
Total number of patients	7	10	1 (1)	6 (1)

[1] Number of patients now dead shown in parentheses.

Partial Resection.—After partial resection it is essential that pleural drainage be adequate, and there must be a top tube for escape of air which accumulates as a result of alveolar leaks after segmental resection or when fissures are incomplete. Suction may be applied to the drainage tubes if the air leak is excessive, or it may be used as a routine measure.

To stop over-expansion of the remaining lung tissue the methods used to reduce the size of the pleural cavity are phrenic nerve crush and thoracoplasty. If thoracoplasty is used, it is best carried out at the time of resection.

After upper lobectomy the usual method of reducing the size of the pleural cavity is by thoracoplasty; occasionally phrenic crush is used.

After lower lobectomy phrenic crush is a routine procedure; pneumoperitoneum may be used in addition.

When no space-reducing measure is employed at the time of resection, it is occasionaliy found that there is incomplete reexpansion of lung tissue and a thoracoplasty is necessary some weeks after resection.

The timing of the thoracoplasty after both pneumonectomy and lobectomy has been the subject of some controversy both within our unit and in other places, and the figures have been analysed to see whether the timing has altered the occurrence of complications.

Pneumonectomy figures are shown in Table VIII.

It will be seen from Table VIII that fistula and empyema and empyema alone occurred three times in the immediate thoracoplasty group, and five times in the combined group of patients who did not have immediate thoracoplasty. On face values this is not a significant difference, but a detailed analysis of the three cases is of interest. One patient had only a five-rib thoracoplasty and a large space remained. One had an emergency pneumonectomy for a spontaneous pneumothorax; the patient was a desperately ill girl, aged fifteen years, and the pleural cavity was secondarily

TABLE X.
Comparison of Results.

Observation.	Austin Hospital: First Series. (100 patients.)	Austin Hospital: Second Series. (100 patients.)	Bickford *et alii*, 1952. (223 patients.)	Zerbeni, 1953. (103 patients.)	Kelly *et alii*, 1952. (101 patients.)	Sive *et alii*, 1952. (84 patients.)	Johansson *et alii*, 1952. (54 patients.)
Patients well	74	—	197	68	82	60	36
Average length of follow-up in months	35	Not done	36[1]	24[1]	16	36[1]	36[1]
Patients dead	15	1	9	25	3	9	9
Early death	7	1	5	13	3	2	7
Late death	8	Not assessed	4	12	0	7	2
Patients with present active disease or invalidism	11	Not assessed	16	10	10	15	7
Not traced	0	Not done	1	0	6	0	2

[1] Not given by authors, but estimated from various figures quoted.

infected heavily before pneumonectomy. The one patient who developed empyema alone was cured by drainage, and the sinus healed; this empyema was a deep-seated localized "stump abscess" and not a general infection of the pleural cavity.

It can thus be stated that no patient who had a "clean" pleural cavity before pneumonectomy and on whom adequate immediate thoracoplasty was performed developed a broncho-pleural fistula or a significant empyema.

Of the 25 patients who had no thoracoplasty, only 22 survived the immediate post-operative weeks, and 21 of them had later thoracoplasty to obliterate the space; one developed a fistula and empyema before thoracoplasty; this operation failed to close the fistula and the patient died.

Lobectomies are reviewed in similar fashion in Table IX.

Examination of this table shows that no fistula and empyema or residual space trouble followed any operation that was accompanied by adequate measures to reduce the space at the time of the resection.

No complications followed the segmental resections in this series.

The statement has been made that no empyema or fistula followed immediate thoracoplasty after resection. It may well be argued that in certain cases collapse prior to resection may be better than immediate thoracoplasty; but here again the figures show that in the 12 such cases fistula and/or empyema occurred three times. After previous thoracoplasty the chest wall has regained its rigidity and is often unable to collapse down to obliterate even the slit-like residual space, with consequent increased danger of empyema. When an immediate and adequate thoracoplasty is performed the soft chest wall is able to come right down onto the remaining lung or mediastinum and effect early obliteration of any residual space.

Comparison with Other Series.

In a reasonably comprehensive survey of the recent literature on the results of resection in the treatment of tuberculosis, the results at the Austin Hospital compare very well with most published series. Unless late complications occur in the second 100 cases, the results in this group will be considerably better than any published to date. However, it is most unwise to attempt to anticipate results in a disease so unpredictable as tuberculosis.

Table X shows a comparison of the two series of patients with five recently published series.

Summary.

The indications for resection in pulmonary tuberculosis have been discussed.

Two series, each of 100 patients, have been analysed, the first in detail, and the second has been briefly reviewed.

Figures have been compared with other recently published series.

Conclusions.

1. In properly selected cases resection offers a very good chance of arrest of disease.

2. Resection is replacing thoracoplasty in the treatment of many patients who in the past would have been treated by this method.

3. Resection aims at removal, as far as possible, of all the diseased lung tissue; but on occasions only the worst areas of disease are removed, with benefit to the patient.

4. In good-risk patients the mortality and morbidity rates are very low (1·8% mortality); in poor-risk patients they are considerably greater (37% mortality), but resection offers the patient the only chance of worth-while survival.

5. The occurrence of broncho-pleural fistula and empyema can be minimized by meticulous bronchial closure with burial of the stump, together with adequate and immediate steps to obliterate the residual space.

Acknowledgements

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ANOMALIES OF THE VITELLO-INTESTINAL DUCT.

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Sydney.

OVER two hundred years before Johann Frederic Meckel published his classical description of the diverticulum which now bears his name, the German surgeon and anatomist Fabricius Hildanus, in 1598, described an intestinal diverticulum. Almost one hundred years later Fredericus Ruysch published the first known picture of a diverticulum, while in 1700 Littré described a hernia which contained an ileal diverticulum. The possibility that this structure might be of congenital origin was first

considered by the Italian pathologist and anatomist Morgagni, who described it as a "true" diverticulum, in contrast to other "false" intestinal diverticula which did not contain all layers of the bowel in their wall. Bose, in 1799, proved that the diverticulum was indeed congenital; but it remained for Meckel, in a series of three classical papers published between 1808 and 1815, to establish that the diverticulum was congenital, solitary and located on the antimesenteric border of the ileum at the site where the vitello-intestinal duct joined the gut (Kittle, Jenkins and Dragstedt, 1947).

DEVELOPMENT.

The vitello-intestinal (omphalo-mesenteric) duct forms a direct communication between the primitive mid-gut and the yolk sac, receiving its blood supply from the two vitelline arteries, branches of the aorta via the superior mesenteric artery. The communication between the gut and the yolk sac narrows gradually and comes to be included in the body stalk until the vitello-intestinal duct undergoes obliteration during the sixth or seventh week of fœtal life (Hamilton, Boyd and Mossman, 1946).

The abnormal persistence of part or all of the vitello-intestinal duct gives rise to the following series of abnormalities.

Persistence of a Patent Vitello-Intestinal Duct.

A history of some abnormality in the cord at birth can usually be obtained. After separation of the cord there is a persistent discharge from the umbilicus, usually mucoid in nature, and an accompanying excoriation of the surrounding skin. There is usually a heaped-up mass of moist granulation tissue at the umbilicus which bleeds easily when traumatized. If fæcal drainage occurs the diagnosis is rendered more obvious, while in those rare cases in which a copious fœcal discharge is present there is usually an atresia of the bowel at a lower level. The introduction of a small amount of "Lipiodol" into the opening and subsequent X-ray examination will often afford positive proof of a direct communication with the bowel.

Once the diagnosis has been established, operation should not be long delayed, because of the high incidence of prolapse of the intestine that occurs through the fistula with the production of an acute obstruction. It is this lethal complication which is responsible for the relatively high mortality associated with this condition. The umbilicus, duct and diverticulum, if present, should be excised and the deficiency in the wall of the ileum carefully repaired so as to produce no stenosis of the gut. In cases in which prolapse has occurred, resection with side-to-side anastomosis of the ileum will usually be necessary unless the prolapse can be reduced, as is sometimes possible shortly after it has taken place.

Cases have been reported in which the vitello-intestinal duct is stated to have opened into the appendix. However, this is not possible, on morphological grounds, and such cases, in which an appendiceal fistula is present at the umbilicus, must have some other explanation (Crymble, 1921; Hadley and Cogswheel, 1936). Jordan reports a case in which the appendix was present in an umbilical hernia and was caught during ligation of the cord with the subsequent production of a fistula.

As in all other anomalies associated with persistence of portion of the vitello-intestinal duct, there is a considerable preponderance in males, for some unknown reason. Kittle *et alii* (1947), in listing 131 known cases of patent vitello-intestinal duct, found that in 93 the subjects were males, in 18 they were females, and in the remainder the sex was not stated.

Vestigial Band Connecting the Ileum to the Umbilicus.

A vestigial band between the ileum and the umbilicus represents either the obliterated vitello-intestinal duct or, less commonly, the vitelline vessels, in which latter case the band joins the mesentery instead of the antimesenteric border of the ileum. It is interesting to note, in this connexion, that rare cases have been recorded in which the patent vitelline vessels have persisted and, in at least one case, ruptured and been responsible for fatal intraperitoneal hæmorrhage (Fraser and McCartney, 1921; Smithy and Chamberlain, 1946).

The band usually dramatically announces its presence when a loop of small bowel becomes kinked over it, an attack of acute intestinal obstruction being precipitated. The sudden occurrence of acute intestinal obstruction in a young child who has previously been quite well and has had no prior abdominal operation should always suggest this diagnosis.

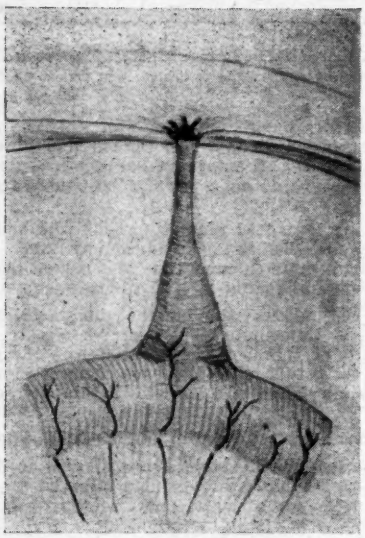

FIGURE I.
Persistence of a patent vitello-intestinal duct.

In other cases the band causes recurrent central abdominal pain of a colicky nature, and the child usually comes to appendicectomy eventually. Unless the terminal portion of the ileum is explored as a routine procedure, the condition will be unrelieved and undetected, and ultimately an acute obstruction is likely to ensue.

Omphalo-mesenteric Cyst.

Omphalo-mesenteric cyst originates in the course of the obliterated vitello-intestinal duct and may retain its attachment to the small bowel, to the umbilicus or to both, or it may form a cyst in the abdominal wall immediately beneath the umbilicus. It is commonly associated with a Meckel's diverticulum, which should be excised with it. Its attachments may cause intestinal obstruction, or the cyst itself may undergo torsion or become secondarily infected. Usually it will be discovered as a smooth, symptomless swelling deep to the umbilicus, and laparotomy will be necessary to discover its true nature.

Umbilical Sinus.

When the umbilical extremity of the duct fails to become obliterated, a number of different abnormalities may be produced. A sinus may be present at the umbilicus which intermittently discharges small amounts of mucus. Secondary infection is common, in which case the discharge will become muco-purulent. There is usually considerable excoriation of the surrounding skin, which will be increased if the discharge contains acid and pepsin, as will be the case if the lining of the sinus includes heterotopic gastric mucosa, as it often does. Such a sinus may be difficult to differentiate from a complete vitello-intestinal fistula; but the instillation of a little "Lipiodol"

should clarify the diagnosis. Such a sinus is frequently attached to the terminal portion of the ileum by a band which represents the remnant of the vitello-intestinal duct or vitelline vessels, and it may be associated with a Meckel's diverticulum, so that its removal should be accompanied by an exploration of the terminal portion of the ileum to deal with any other associated anomaly.

Eversion of the unobliterated umbilical extremity of the duct will give rise to the typical "raspberry tumour" of the umbilicus which, once again, may be associated with a band between the umbilicus and ileum and/or a Meckel's diverticulum. Acid-secreting cells may be present in the

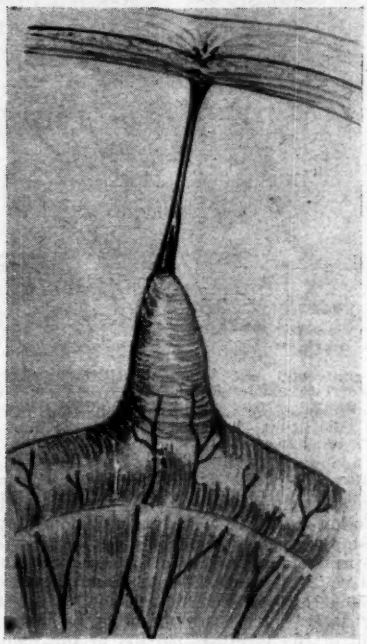

FIGURE II.

Vestigial band connecting the apex of a Meckel's diverticulum with the umbilicus. In other cases, such a band connects directly with the ileum or, if it represents the vitelline artery, with the mesentery of the terminal portion of the ileum. The umbilical end may be free or may become reattached to the mesentery or posterior abdominal wall.

umbilical polypus. It is best dealt with by diathermy excision, and at the same time the terminal portion of the ileum should be explored. The term "enteroteratoma" sometimes used to describe this condition is, of course, a misnomer and should be discarded.

Stenosis of the Terminal Portion of the Ileum.

It is possible that in some cases ileal stenosis occurring within five feet of the ileo-caecal valve is due to excessive involution of the vitello-intestinal duct. The condition may never give rise to any symptoms; but it can be the site of impaction of a foreign body or, if extreme, will give rise to a low small-bowel obstruction of greater or lesser degree. There is no way of distinguishing this condition from other cases of ileal stenosis or atresia which are due to a failure of recanalization of the gut. Treatment consists of entero-anastomosis above and below the lesion.

Meckel's Diverticulum.

Meckel's diverticulum, an outpocketing of the terminal portion of the ileum, is present in approximately 2% of the population according to post-mortem figures. In many cases its presence causes no inconvenience to its possessor, but it may give rise to a variety of serious lesions, as listed below. It most commonly causes trouble in childhood; but there are numerous cases on record in which it has not produced any serious incident until well into adult life. However, 90% of subjects in whom symptoms appear are aged under thirty years, and most are aged under fifteen years.

Since Meckel's diverticulum represents the unobliterated intestinal end of the vitello-intestinal duct, the following criteria must be fulfilled before a diverticulum can be classified as a member of this group: (i) It must be

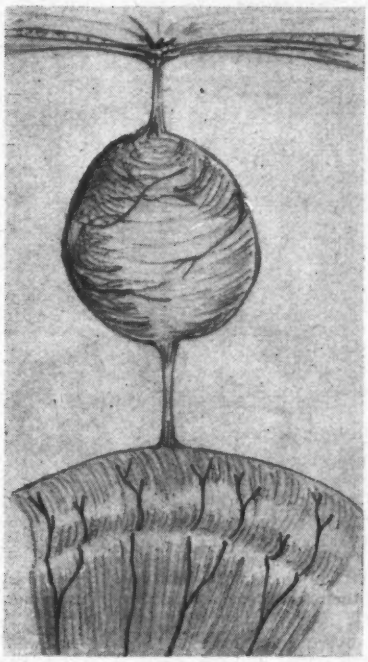

FIGURE III.

Omphalo-mesenteric cyst. This may be attached to the umbilicus or the terminal portion of the ileum or both. A Meckel's diverticulum may be present as well. The cyst is sometimes situated in the abdominal wall deep to the umbilicus.

situated on, or near to, the mesenteric border of the ileum. (ii) It must have its own separate blood supply. (iii) It must contain all layers of the small bowel.

It is commonly taught that the diverticulum is always within three feet of the ileo-caecal valve. However, in a recent review of 103 cases, Jay and his colleagues (1950) found that in only 72% was it within this distance; in the remaining 28% it was up to five feet from the ileo-caecal junction.

There is considerable variation in the size and shape of the diverticulum. The majority of diverticula are under five centimetres in length, but the longest reported which fulfils the criteria laid down above was 95 centimetres in length (Jay *et alii*, 1950). Other longer "diverticula" which have been reported appear in most cases to have been duplications of the bowel. In most cases the diverticulum is short, smooth-walled and wide-based. However, secondary diverticula are not uncommon, and one of these is not infrequently the site of perforation. The method of junction of the diverticulum with the ileum is also subject to considerable variation, so that one method of removal will not be applicable to all cases. Figure VI shows some of the different appearances that may be seen.

The tip of the diverticulum may be free or may, less commonly, be attached to the umbilicus by a band. This band may, on occasions, lose its attachment to the umbilicus and either remain free or reattach itself to the posterior abdominal wall, to the mesentery or to a loop of small bowel in the vicinity.

The diverticulum is lined by mucosa similar in nature to that in the ileum; but heterotopic epithelium of either gastric or pancreatic type is frequently present. Mucosa identical in type with that present in the duodenum, jejunum, colon and bile ducts has also been described. Any

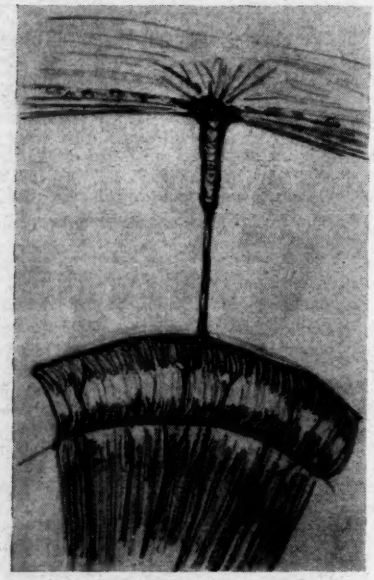

FIGURE IV.

Umbilical sinus. This may, or may not, be attached to the terminal portion of the ileum by a band. A Meckel's diverticulum may be present as well. Eversion of this sinus produces the so-called "raspberry tumour" of the umbilicus.

type of gastric epithelium may be present, and both hydrochloric acid and pepsin may be secreted by the diverticulum (Curd, 1936; Matt and Timpone, 1940). The presence of gastric mucosa in the diverticulum renders it much more likely to undergo pathological changes. In diverticula which are discovered *post mortem*, or incidentally at operation—diverticula which have never been productive of symptoms—the incidence of heterotopic gastric mucosa is about 20%. Of diverticula which are productive of symptoms, however, over 60% will be found to contain gastric mucosa (Jay *et alii*, 1950). These figures vary from centre to centre, depending on the methods employed to detect heterotopic mucosa. If serial sections are used the figure will be higher than if only one or two sections through the diverticulum are examined. Since the former method is laborious and time-consuming, it is not as a rule employed, and therefore most figures tend to under-estimate the number of diverticula which do in fact contain gastric mucosa.

The following pathological changes have been found to occur in Meckel's diverticula.

Inflammation.

Acute diverticulitis is productive of symptoms which are indistinguishable from those of acute appendicitis. The demonstration of a normal appendix under such circumstances should prompt the surgeon immediately to explore the terminal five feet of ileum to exclude this lethal condition. Should an acutely inflamed diverticulum rupture, and this is frequently the case, localization is unlikely to occur and spreading peritonitis is the rule.

Chronic inflammation can also occur. This may be specific — tuberculous diverticulitis has been recorded (Macdonald, 1947; Michael, 1932)—or non-specific, possibly owing to the presence of gastric mucosa, and results in peri-diverticulitis with the development of adhesions between the diverticulum and the neighbouring bowel, mesentery or omentum. Clinically this results in vague, recurring central abdominal pains sometimes associated

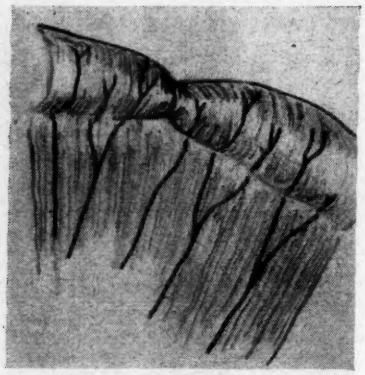

FIGURE V.

Stenosis of the terminal portion of the ileum. This may possibly be due to hyperinvolution of the vitello-intestinal duct.

with deep tenderness to the right of the umbilicus. A barium "follow-through" X-ray examination is of very little help in establishing the diagnosis. It is most exceptional to be able to demonstrate the diverticulum in this manner, and negative X-ray findings are of no significance whatsoever. Most of these children come to appendicectomy sooner or later. It is my practice to make a routine exploration of the terminal portion of the ileum in every case in which appendicectomy is performed unless the acuteness of the local lesion makes this procedure inadvisable. It is very disconcerting to have a patient return after appendicectomy with persistence or recurrence of the pain that was present pre-operatively—and this does occur on occasions—and not to have excluded a Meckel's diverticulum. This routine exploration of the terminal portion of the ileum, if gently performed, is not productive of any ill effects. I have made it on more than 200 occasions without incident, and it adds very little to the duration of the operative procedure. It is preferable to examine four to five feet of ileum, as otherwise a percentage of diverticula will be overlooked. The following two case histories are illustrative of the value of this routine procedure.

CASE I.—A, aged five years, a female patient, had suffered from recurrent umbilical pain lasting up to an hour for the past six months. On the day of her admission to hospital periumbilical pain had suddenly commenced immediately after the child had vomited, and the pain had persisted. On examination of the patient, the tongue was dry and furred and the breath offensive. Lower abdominal tenderness and guarding were present, most pronounced in the right iliac fossa. A diagnosis of acute appendicitis was made. The abdomen was opened through a McBurney's incision. Free fluid was present, and the appendix was located in a retro-caecal position. It did not appear abnormal on macroscopic examination. A Meckel's diverticulum was found two feet from the ileo-caecal valve, which was adherent to the mesentery. It was dissected free and removed, and appendicectomy was performed. Convalescence was uneventful. The pathologist reported the presence of small acute inflammatory foci in the mucosa of the appendix. The Meckel's diverticulum showed no evidence of recent inflammation and did not contain gastric mucosa.

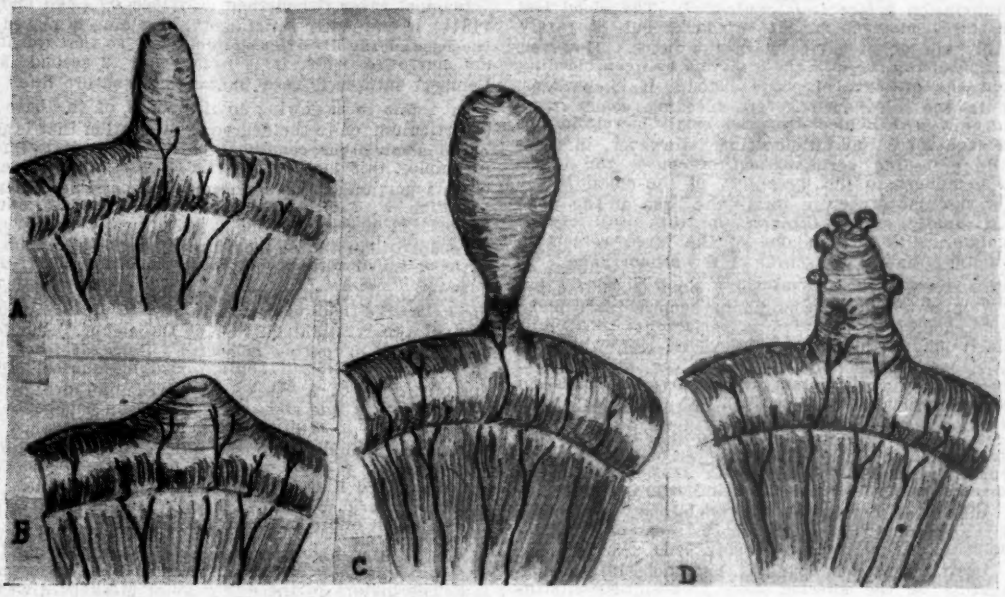

FIGURE VI.

Meckel's diverticulum. A: The type that is most commonly found. B: Short, broad-based type that is occasionally seen; it is not possible to remove this variety except by resection. C: Narrow-based diverticulum; this type is most likely to undergo torsion. D: Secondary diverticula on a Meckel's diverticulum. These are not uncommon.

CASE II.—B, aged six years, a male patient, had a history of recurrent periumbilical pain present intermittently for twelve months. Occasional vomiting had occurred with the attacks of pain. He was admitted for routine appendicectomy. The abdomen was explored through a McBurney's incision and a retrocaecal appendix was removed. Routine exploration of the terminal portion of the ileum revealed a Meckel's diverticulum situated three feet from the ileo-caecal junction. The diverticulum was attached to the umbilicus by a band which was twisted, owing to the rotation of the loop of ileum to which it was attached. The umbilical attachment of the band was severed and diverticulectomy was performed. Convalescence was uneventful. The pathologist reported that the appendix was normal on microscopic examination and that the diverticulum contained gastric mucosa.

The presence of heterotopic gastric epithelium renders a Meckel's diverticulum peculiarly liable to the occurrence of peptic ulceration, usually at an early age. Postprandial discomfort and pain, coming on an hour or more after a meal and referred to the periumbilical region, accompanied by local tenderness, are the usual findings. However, one is not long left in doubt as to the diagnosis, for as a rule hæmorrhage or perforation will occur.

Perforation comes on with dramatic suddenness and gives all the signs of a spreading peritonitis. If a preceding history of dyspepsia of characteristic type is obtained, it may suggest the true nature of the perforation. Treatment consists of laparotomy and diverticulectomy together with suitable supportive therapy to correct the fluid balance and combat infection. Even under modern conditions this serious complication carries an appreciable mortality, and prior to the advent of the antibiotics and modern methods of fluid replacement, it caused the death of the patient in more than 50% of cases.

Hæmorrhage from an ulcer in a diverticulum is often severe and recurrent, and in many cases will necessitate

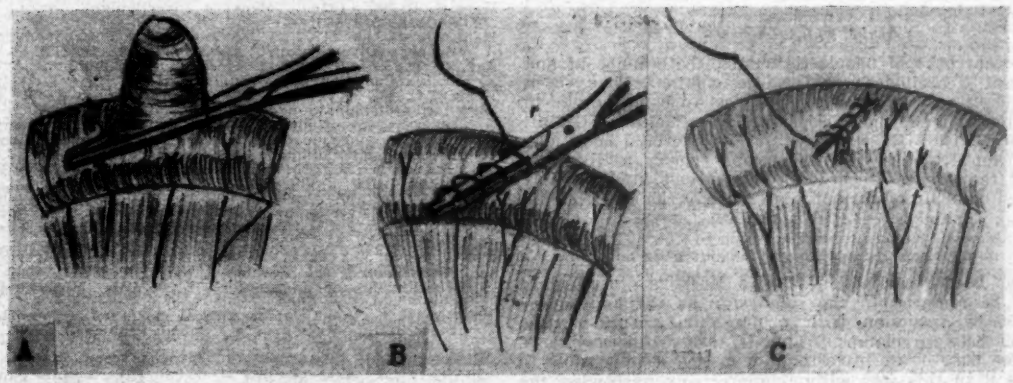

FIGURE VII.

Method of removing a Meckel's diverticulum (after Ladd and Gross, 1941). The base of the diverticulum is clamped obliquely to avoid narrowing the gut, and removed between clamps. The remaining clamp is oversewn and removed and the suture line is reinforced with a layer of Lembert sutures.

blood transfusion before it is controlled. The blood lost in such cases may be bright or dark, but is rarely appreciably altered or mixed with the fæces. However, examination of the fæces in the intervals between bleeding will show the presence of occult blood. It is, perhaps, difficult to see why such severe bleeding comes from these ulcers. Other ulcers of the small bowel do not provoke such torrential hæmorrhage. However, in these cases the preceding peridiverticulitis causes the diverticulum to adhere to the mesentery of the small bowel, and this vascular structure forms the base of the ulcer when it rapidly erodes through the thin wall of the diverticulum, so that it is one of the large vessels in the mesentery that is the source of the hæmorrhage.

In other cases an occasional tarry stool is passed, but sooner or later a severe hæmorrhage usually takes place. The occurrence of severe bleeding *per rectum* in a child, particularly under the age of five years, for which no obvious cause can be found, makes the diagnosis of bleeding Meckel's diverticulum presumptive and calls for laparotomy as soon as the condition of the child allows this to be carried out with safety.

Obstruction.

In most cases of obstruction a band running from the tip of the diverticulum to the umbilicus, mesentery, posterior abdominal wall or a nearby loop of bowel is the responsible agent, as has already been described. A simple obstruction may result, but not infrequently strangulation of the affected loop occurs, necessitating resection, which carries an appreciably higher mortality in childhood, particularly in the younger age group.

The usual story is that of a sudden acute small bowel obstruction in a child who has previously been quite well and has had no previous abdominal operation.

Intussusception.

It is well known that an inverted Meckel's diverticulum may form the leading point of an intussusception, particularly in children over the age of two years. In this age group it is essential to explore the terminal portion of the ileum to exclude this abnormality, even if a high saline or barium enema has been used to reduce the intussusception. Indeed, many surgeons consider that this should be done in all cases. If the condition of the child permits, the diverticulum should be resected at the time when it is discovered. One finds in practice that many of these children have an irreducible intussusception, so that resection is necessary (Harkins, 1933).

Torsion.

Torsion is an uncommon complication of a Meckel's diverticulum which may result in gangrene or rupture of the diverticulum with spreading peritonitis (Poate, 1935).

Other Complications.

The occurrence of neoplastic change, both innocent and malignant, carcinomatous or sarcomatous, has been recorded on several occasions. The change may occur in the heterotopic epithelium of the diverticulum. Calculi may form within the diverticulum or a foreign body may be entrapped within it and possibly perforate its wall. One of the earliest recorded complications, and possibly the least common of all, is the presence of the diverticulum within a hernial sac—Littré's hernia.

Removal of Meckel's Diverticulum.

It has been recommended that a Meckel's diverticulum should be removed in the same way as the appendix—that is, by inversion with a purse-string suture after excision between clamps. This is rarely practicable, as the wide base of the diverticulum makes it impossible to remove it in this way without appreciably narrowing the ileum in most cases. Even in those uncommon cases in which the base is narrow enough to permit of the use of this method, the inverted stump might well form the leading point of an intussusception.

In most cases the method described by Ladd and Gross (1941) is eminently satisfactory. A clamp is placed across the base of the diverticulum obliquely so that the ileum is not narrowed when it is oversewn. A second layer of Lembert sutures is used to invert the suture line.

In a few cases, owing to the width of the base of the diverticulum, or to the degree of induration that is present, or to some other complicating factor, it is not possible to employ this method. In these cases a wedge excision of this portion of the ileum may be possible, otherwise a resection with either an end-to-end or side-to-side anastomosis is necessary. This is naturally a more major procedure than the method already outlined, which causes little or no disturbance of bowel function as a rule.

Finally, it is necessary to consider the symptomless Meckel's diverticulum that is discovered on routine exploration of the terminal portion of the ileum. Should it be removed as a routine measure? Since at least 20% of these diverticula will contain gastric mucosa, since the complications of a Meckel's diverticulum are of such a serious nature, and since its removal can usually be accomplished with a minimal amount of disturbance to the patient, it should be excised unless the width of its base would render a bowel resection necessary for its removal, in which case it is thought that it should be left and its presence noted so that it may be sought at a later date if symptoms of its presence develop.

SUMMARY.

1. The history of the discovery of Meckel's diverticulum and the embryology of the vitello-intestinal duct are outlined.

2. The abnormalities associated with the persistence of part or all of the vitello-intestinal duct are listed and discussed.

3. Meckel's diverticulum is fully discussed, including its complications and methods of removal.

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ACUTE POST-PARTUM RENAL FAILURE (LOWER NEPHRON NEPHROSIS), WITH A REPORT OF TWO UNUSUAL CASES.

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THE term "lower nephron nephrosis" was introduced by Lucke (1946) to designate the syndrome of anuria or oliguria with uræmia, degenerative changes and necrosis in the distal convoluted tubules, and deposition of pigmented casts in the distal and collecting tubules. The syndrome followed intravascular hæmolytic reactions, crushing injuries to muscles, burns, sulphonamide intoxications, toxæmia of pregnancy, heat stroke, severe alkalosis and post-abortional states. After reviewing 538 fatal cases Lucke concluded that the fundamental factor was renal ischæmia. On pathological examination the kidneys are swollen and heavier than normal, and have a pale cortex and dark medulla. On microscopic examination the principal lesions are focal degeneration, vacuolization, desquamation and necrosis of the epithelium of Henle's loop and the distal segments of the tubules, while the glomeruli and proximal tubules are not severely disturbed (Goormaghtigh, 1945). Pigment casts can be seen in the lower nephrons and collecting tubules, the pigment being myoglobin in cases of "crush syndrome" and hæmoglobin in cases of intravascular hæmolysis. It is now considered that the presence of pigmented casts is a secondary effect of anuria (Corcoran and Page, 1947; Young, 1942), and the underlying mechanism is vasoconstrictive renal ischæmia.

The actual "mechanism" referred to above was first demonstrated by Trueta, Franklin, Barclay and Daniel (1946), who, after experimenting on animals, showed that the stimulation of renal vasomotor nerves resulted in a redistribution of renal blood flow, so that the cortex which normally received most of the flow became ischæmic, while the main stream was diverted to the medulla. They concluded that the "shunt mechanism" was primarily protective to exclude the cortex from circulating toxins and noxious agents, but that too prolonged operation of the device resulted in permanent kidney damage. Thus the "Oxford shunt" may underlie the pathological changes found in crush syndrome and allied states.

In obstetric practice, oliguria is usually associated with abortion, toxæmia of late pregnancy (particularly when complicated by accidental hæmorrhage) and transfusion of incompatible blood. Lower nephron nephrosis and bilateral symmetrical cortical necrosis are the main pathological conditions found in these cases. Occasionally, urinary obstructions from sulphonamide deposition and "reflex" anuria have been observed (Gill, 1951).

Lower nephron nephrosis may complicate any period of gestation (O'Donnell, 1949). It has been observed in the following circumstances: in post-abortional states; after the use of abortifacient drugs and soap solutions; in infections with bacteria which produce hæmolytic toxins (*Clostridium welchii, Staphylococcus aureus*) (Bull, Joekes and Lowe, 1949); after incompatible blood transfusions; in sulphonamide intoxication; in severe hæmorrhage or shock. It has also been associated with abortion in the absence of shock (O'Donnell), possibly in a way analogous to the occurrence in crush syndrome, the placental debris being equivalent to devitalized tissue (Young, 1942).

The syndrome has occurred in pregnancy associated with accidents of labour (Young and McMichael, 1941), and with concealed accidental hæmorrhage (Mauzy and Donnelley, 1949; Young, 1941). Paxson, Golub and Hunter (1946) have also reported their experiences with the syndrome accompanying accidental hæmorrhage. Two cases of lower nephron nephrosis in the immediate post-partum period are described below.

Reports of Cases.

CASE I.—Mrs. A., a *primigravida*, aged thirty-four years, was first examined at the ante-natal clinic at St. Mary Abbot's Hospital on July 30, 1951, at the thirty-sixth week of her pregnancy. She felt well. The blood pressure was 120 millimetres of mercury, systolic, and 75 millimetres, diastolic. No œdema was present. The urine was free from albumin. There was no abnormality of heart or lungs. The uterus was the size of a thirty-six weeks' gestation. The hæmoglobin value was 10·8 grammes *per centum*. The Wassermann and Kahn tests produced negative results. Her blood group was O, Rhesus-positive. X-ray examination revealed a normal single fœtus, presenting by the vertex.

The patient was subsequently examined on three occasions at weekly intervals, and no abnormality was found. On August 30, 1951, the patient went into labour spontaneously and was delivered of a still-born male fœtus, weighing six pounds three ounces. (Subsequent post-mortem examination revealed changes due to asphyxia.) The first stage lasted fifteen hours fifty minutes; the second stage lasted thirty minutes; the third stage lasted five minutes. There was a post-partum hæmorrhage of about 852 mils (513 mils being lost before, and during, the delivery of the placenta, and 339 mils half an hour afterwards). The cause of the post-partum loss was uterine inertia, and after the administration of ergometrine (0·5 milligramme given by intramuscular injection and 0·125 milligramme by intravenous injection) there was no further bleeding. The placenta was normal, battledore in type, and was complete. After delivery the patient's blood pressure was 95 millimetres of mercury, systolic, and 70 millimetres, diastolic. Her temperature was 99° F. and her pulse rate 88 per minute. After the second blood loss (339 mils) the patient showed signs of severe shock; the blood pressure dropped to 75 millimetres of mercury, systolic, and 50 millimetres, diastolic. A transfusion was promptly given (568 mils of dextran, followed by 1136 mils of blood). The shocked state lasted two hours. The patient was oliguric from the start. Vomiting occurred once. On the third day the patient became drowsy and non-cooperative. The blood pressure was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic; the temperature was 98·4° F., and the pulse rate 80 per minute. There was no œdema or jaundice. The spleen was not palpable. The specific gravity of the urine was 1·006. Albumin was present. Microscopic examination revealed occasional pus cells, many round and pear-shaped epithelial cells, and red blood cells. Biochemical examination of the urine showed it to contain 0·06 milligramme of protein per 100 mils, 340 milligrammes of chlorides per 100 mils, and 0·4 gramme *per centum* of urea. Blood examination gave the following information: the hæmoglobin value was 7·7 grammes *per centum*, the urea content was 180 milligrammes *per centum*, and the chloride content was 460 milligrammes *per centum*. The serum sodium content was 300 milligrammes per 100 mils, and the serum potassium content 23 milligrammes per 100 mils. The alkali reserve was reduced to 35·4 volumes of carbon dioxide *per centum*.

A diagnosis was made of lower nephron nephrosis following post-partum hæmorrhage and obstetric shock.

Treatment on conservative lines (as recommended by Bull, Joekes and Lowe) was commenced early in the anuric phase. A high caloric diet (2000 Calories) free from protein was given until the tenth day. Fluid administration was limited to a basic intake of one litre daily. No electrolytes were given. On the tenth day when diuresis was established a light protein diet was allowed, and the fluid intake was gradually increased. A blood transfusion was given on the eleventh day. There was a period of anuria, followed by a period of oliguria during the first five days. (See Figure I.)

Diuresis commenced on the seventh day and was maximal on the tenth day. Albuminuria disappeared after the tenth day. The urinary urea output increased after diuresis was established. The blood urea content rose to 265 milligrammes per 100 mils by the tenth day and then fell quickly. The serum chloride values did not vary outside the limits of normal. There was slight acidosis during the anuric and oliguric phases. The serum potassium and sodium values were estimated on the first, eleventh and fifteenth days and were within normal limits on each occasion. When she was discharged from hospital on the twenty-eighth day, the patient felt well. The blood urea content was 30 milligrammes per 100 mils. The specific gravity of the urine was 1·008, and no albumin was present. Microscopic examination revealed occasional epithelial cells and leucocytes. The protein content of the urine was nil; the chloride content was 265 milligrammes per 100 mils. The urea content was 0·759 gramme *per centum*.

CASE II.—Mrs. B., aged thirty-seven years, pregnant for the ninth time, was admitted to Chiswick Hospital on April

1, 1953, with the diagnosis of obstructed labour. The antenatal period had been normal. Labour had commenced fourteen hours before her admission to hospital. When there had been no advance in labour after the second stage had lasted three hours, the midwife called for medical aid and the patient was sent to hospital. On her admission, her temperature was 99·2° F., her pulse rate was 80 per minute, and her respirations numbered 20 per minute. Her blood pressure was 112 millimetres of mercury, systolic, and 72 millimetres, diastolic. No œdema was present. The urine contained a trace of albumin. The tongue was moist and clean, the chest and heart were normal, and there was no abdominal tenderness. The uterus was at term, contracting at five to ten minute intervals, and the fœtus was lying longitudinally, its head in the pelvic cavity of the mother. Vaginal examination revealed a face presentation, the fœtus being in the mento-anterior position. The patient was pre-

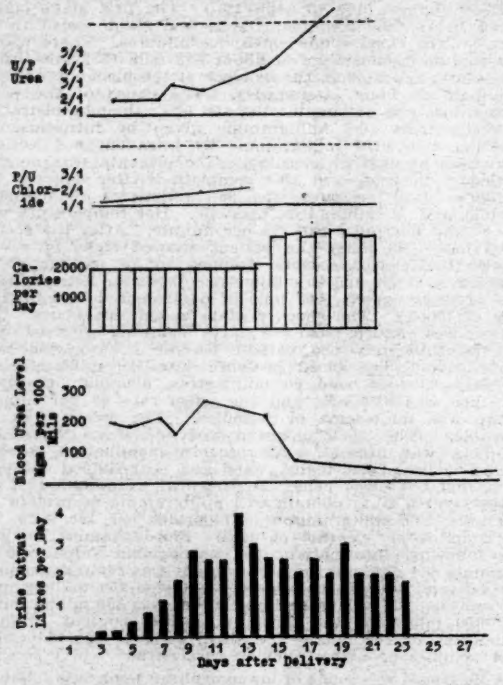

FIGURE I.

U/P urea = urine concentration divided by plasma concentration. P/U chloride = plasma concentration divided by urine concentration. (In both cases, a ratio of over 5:1 indicates satisfactory return of tubule function.) Open columns, Calorie intake; solid columns, urine volume.

medicated with atropine, and anæsthesia was induced with "Sodium Pentothal" (0·5 gramme given intravenously) and maintained with cyclopropane and oxygen. A low application of forceps was performed, the shoulder was disengaged and a still-born male anencephalic fœtus was delivered. It was macerated and weighed seven pounds 10 ounces. The placenta was delivered spontaneously after five minutes; it weighed one and a quarter pounds and was normal and complete. The total blood loss was 120 mils. After delivery the patient's condition was satisfactory. Her blood pressure was 110 millimetres of mercury, systolic, and 72 millimetres, diastolic, her pulse rate was 80 per minute, and her temperature was 99·4° F. Eighteen hours after delivery, the patient became jaundiced and developed hæmoglobinuria. Examination revealed that her temperature was 98·4° F., and her pulse rate 88 per minute. The tongue was moist and coated with white fur. The abdomen was lax and not distended, and moved with respiration. Neither the liver nor the spleen was palpable. The uterus lay obliquely to the left, and felt tender. The lochia was red and non-offensive. There was no œdema, ecchymosis or bruising. The hæmoglobin value was 10 grammes *per centum*, and the red cells numbered 5,290,000 per cubic millimetre. The mean corpuscular hæmoglobin was 30γγ. The white cells numbered 47,000 per cubic millimetre (6% were lymphocytes, 90% polymorphonuclear cells and 2% monocytes). The fragility of the red cells was within normal limits. The Wassermann test produced a negative result, and an attempt at blood culture gave negative results. *Clostridium welchii* organisms were grown on culture from the vaginal swab. The patient was isolated and penicillin therapy was begun. The urine was alkalinized. During the succeeding twenty-four hours the jaundice deepened and the patient vomited frequently. Oliguria set in on the third day, when the total daily urine output was 106·5 mils. On the fourth day only 7·1 mils of urine were obtained by catheterization. The blood urea content was 201 milligrammes per 100 mils.

A diagnosis was made of lower nephron nephrosis following intravascular hæmolysis of unknown origin.

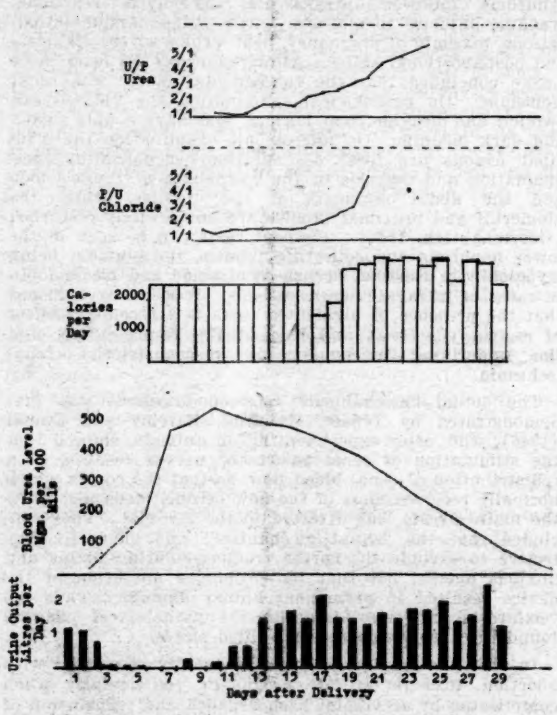

FIGURE II.

U/P urea = urine concentration divided by plasma concentration. P/U chloride = plasma concentration divided by urine concentration. (In both cases, a ratio of over 5:1 indicates satisfactory return of tubule function.) Open columns, Calorie intake; solid columns, urine volume.

The patient was treated conservatively by the Hammersmith Team with a modified Boerst diet during the oliguric phases, with careful water and electrolyte balance, and subsequently with a diet of low protein content in the early diuretic phase. Blood transfusion was required in the convalescent period. Spontaneous diuresis occurred (one litre of urine being excreted) on the thirteenth day. (See Figure II.)

Comment.

Two interesting cases of acute renal failure in the postpartum period were observed. In each instance lower nephron nephrosis occurred after a complicated delivery.

In Case I it seems likely that the vasoconstrictors of severe shock acted on the renal circulation (in a manner similar to intense vasomotor stimulation) and caused cortical ischæmia, which was prolonged and resulted in renal damage.

In Case II severe intravascular hæmolysis preceded the onset of anuria. Mason and Mann (1931) have shown

that renal vessels will tolerate a certain amount of free hæmoglobin in the blood-stream, but when the amount is increased above a certain threshold they respond with contraction. This may have been the mechanism underlying the renal ischæmic damage in this instance.

The history, examination and measurement of the urine, and the blood urea estimation, were the chief factors in establishing the diagnosis.

The conservative *régime* of treatment recommended by Bull, Joekes and Lowe was instituted immediately in the anuric phase. The chemistry of the blood and urine were investigated daily.

Both patients responded favourably to treatment. Diuresis occurred on the seventh day in Case I, and on the thirteenth day in Case II, and in each instance was preceded by phases of anuria and oliguria.

Summary and Conclusions.

1. Two cases of lower nephron nephrosis occurring in the immediate post-partum period are described and discussed.

2. Conservative treatment, instituted early in the anuric phase, was successful in both cases.

3. Accurate urine measurement is important after complicated deliveries.

Acknowledgements.

I wish to thank Dr. D. A. D. Davies, M.D., M.R.C.O.G., and Dr. Amy Fleming, F.R.C.O.G., for permission to describe their cases, and Dr. G. M. Bull, M.R.C.P., for permission to reproduce his chart.

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TREATMENT OF SCHIZOPHRENIA BY COMBINED BARBITURATE-SUCCINATE THERAPY.

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IN recent years various attempts have been made to replace the classical treatment of psychotic conditions (namely, deep insulin or electroconvulsive coma) by somewhat milder procedures—for example, electronarcosis (Frostig *et alii*, 1944), carbon dioxide therapy (Meduna, 1950), narcotherapy (Brickner *et alii*, 1950), and other related procedures. Zeleva (1953) used barbiturate followed by caffeine. Other Russian workers proceeded on similar lines, using sleep therapy combined with "Pyramidon", "Cardiazol", amphetamine or glutamic acid. The original publications unfortunately were not accessible to us, and the abstracts do not clarify to what extent the procedures were intended either as a therapeutic index or as a tentative therapy combining the opposing effects of barbiturates and analeptics. In this latter context we should like to present the histories of a few cases in which mild coma was induced by the intravenous injection of "Pentothal", followed by succinate, to the extent of a total of 20 to 40 treatments; the after-treatment consisted in each case of sodium succinate given orally.

The following procedure was usually adopted. The patient was given a course of sodium succinate tablets, one gramme three times a day, for two or three days. Then he was given, about two hours before lunch, a slow intravenous injection of 5% sodium succinate solution buffered to pH 7·0, in amounts which were just able to produce the characteristic cough and flush (Barrett, 1947; Trautner *et alii*, 1953); usually the quantity was 20 to 30 millilitres. The quantity subsequently injected was higher (40 to 60 millilitres). It was preceded by an injection of atropine, 0·5 milligramme, followed in half an hour by "Pentothal Sodium" given intravenously in a quantity sufficient to produce about thirty minutes of deep sleep, 0·5 to 0·6 gramme being required. When the sleep began to lighten, but before the patient was fully awake, the previously established quantity of succinate was slowly injected. Most patients awoke during the injection, but were inclined to relapse into a pleasant sort of drowsiness which lasted for thirty to forty minutes. No attempts were made either to rouse the patients or to involve them in any probing conversation. The patients did not manifest any signs of agitation. Occasionally they exhibited a state of mild confusion associated with the production of emotionally charged conversation. More frequently they merely stated that they had had a beautiful sleep and regretted that they could not achieve the same sleep during the night. This barbiturate-succinate routine was repeated five times per week until a distinct improvement was observed. Then barbiturate was given every second day and finally stopped altogether; the succinate injections alone were continued for a total of 30 to 50 injections. Thereafter, the patient received succinate tablets only during the following months; then the dose was slowly reduced till an optimal maintenance dose was reached.

Reports of Cases.

CASE V.—A science graduate, Mr. X., aged thirty-two years, had been working until a few months earlier. There was no previous history of mental disturbance. The first symptom, observed about a year previously, was that his letters were becoming incoherent and rambling; then he began to neglect his work and concentrate on inventions. He developed grandiose ideas about going abroad and lecturing, and he became more and more restless; he would not sleep and spent most of his nights on his inventions. After being interviewed by a doctor, he said: "Professor A. was also there; I didn't see him, but I heard his voice." He received several electroconvulsive treatments without benefit and refused to continue the treatment. He became increasingly difficult; he refused to eat, sleep, take medicine or move from any place where he had decided to stay, even to the extent of becoming violent. Once he had to be forcibly removed from a lift.

When first examined the patient was initially mistrusting and antagonistic, but he soon relaxed and began to talk about his inventions, about a conflict situation between his family, himself and his fiancée, and about his uncertain financial position and prospects—all in a desultory way with sweeping gestures of his arms. "One side says this, the other that. What does it matter what I say?" He was found to be bodily healthy and of strong muscular build. He received 15 full coma insulin treatments without benefit and showed increasing resistance to the treatment. He was then given barbiturate-succinate and improved after a few injections. Altogether he received 10 barbiturate treatments and 25 succinate injections. He was released in June, 1953, with a maintenance dosage of five tablets of sodium succinate, each of 0·5 gramme, per day. A few weeks afterwards he returned to work. He was found to be quite free from emotional conflicts. When last examined, in January, 1954, he was still taking three tablets per day and was leading a normal, active life and intended to marry soon.

CASE VII.—Y., a male orphan, aged twenty-eight years, had been brought up by a guardian and was living with her. The guardian stated that the patient always was a quiet child, with normal, healthy interests, but that during the last few months he had become more and more withdrawn and depressed until finally he was quite unable to continue as an accountant—the position he had occupied up till then. On his admission to hospital in March, 1953, he was found to be healthy and strongly built; but he was unresponsive in conversation, tearful, and probably suffering from aural and visual hallucinations. It was not possible to extract from him more than a few moans, weepings and meaningless monosyllables. A course of full coma insulin therapy (31 comas, totalling twenty hours) was completed in May, 1953, without benefit. After each coma the patient would lie in bed for six to eight hours, incontinent of urine and fæces, with tears rolling down his cheeks and occasional meaningless utterings, unable to stand on his legs without support while the sheets were changed. He received his first succinate injection during the post-comatose confusional state in one of his last insulin treatments. Within five minutes his mind cleared and he was able to get out of bed, though unsteadily, to go to the shower room. After completion of his insulin treatment he was catatonic and completely anergic, incontinent of urine and fæces, and unable to dress or wash himself; he had to be spoon-fed. In June he received six electroconvulsive treatments together with intravenous succinate injections, but he showed only slight improvement. Then a course of eight barbiturate-succinate treatments was given, at the end of which he had improved to the extent of being able to look after himself in all respects of toilet, clothing and feeding; he began to talk intelligently, though hesitantly, and to attend occupational therapy classes. He was given 25 succinate injections altogether, and when he was released in August, 1953, his state of improvement was maintained by administration of one gramme of sodium succinate three times a day. His condition further improved slowly, and he began to work on a farm in October. When last examined, in January, 1954, he appeared to be fully recovered and was looking for a position in his old profession as an accountant. He is still taking three tablets of sodium succinate per day.

CASE XI.—A., a strong, healthy boy looking considerably older than his sixteen years, was admitted to hospital in February, 1953, in an acutely excited condition. He was very noisy and inclined to be violent; at times he imagined he was a biblical king, at other times he was just noisy, silly and destructive. Full coma insulin therapy was started in March and continued to the extent of 35 treatments and twenty hours of coma, but without any benefit. After each coma the patient would be "fighting mad" for several hours, breaking chairs and windows and tearing the bedclothes to pieces, or else he would crawl on all fours, yelling and barking. Succinate given intravenously was tried during the last few post-coma periods of confusion, and cleared them within a few minutes. At this stage, the patient showed no improvement in his mental state, but rather a deterioration to the extent of complete disregard for his toilet and clothes; he ate like an animal (with both hands in his food), and it was impossible to maintain any sort of conversation with him. In May he received 15 barbiturate-succinate treatments followed by 10 succinate injections only, and he then showed considerable improvement in so far as he was no longer noisy and violent and generally less agitated; but he still required sedation every night. He attended occupational therapy classes and was transferred to a convalescent ward. Shortly afterwards he was taken home by his family and continued with oral sodium succinate therapy, three grammes per day. During the first few weeks after his release he did nothing, but then he suddenly began to devote a good deal of attention to his hitherto entirely neglected teeth and his wardrobe. In August he started part-time work as a commercial traveller, and in October full-time work in a store. When last examined, in December, 1953, he had just passed his entrance examination as an engineering apprentice with a large firm, had joined a football club, and enjoyed weekly hikes and similar entertainments very much. He is still taking three tablets of sodium succinate per day.

CASE XII.—B., aged twenty-nine years, a farmer's son, had been ill for the last two years. The certificate accompanying the request for admission to hospital stated that he was emotionally flat and oblivious of his surroundings, occasionally breaking out into fits of laughter, and that he admitted to humorous auditory hallucinations as the cause of this laughter. When first examined in July, 1953, he was bodily fit, though rather delicate for a farmer; he was lying in bed with no intention of getting up, with an amused expression on his face, and was addressing jocular remarks to anybody near him. He was never violent and showed no evidence of any more profound emotions or apprehensions. To conduct any sort of coherent intelligent conversation with him was impossible. When asked why he stayed in bed or sat in the dark instead of working, he replied cheerfully that all his strength was gone. Treatment was commenced with succinate given by mouth, two tablets three times a day, and with a course of 10 barbiturate-succinate injections, followed by 20 injections of succinate only. After the first injection he stated that his brain felt clearer and that "less of the thoughts were coming into his head". The first step towards improvement apparently was the loss of his previous auditory hallucinations; he also began to take some intelligent interest in his surroundings. A few days later he began to attend occupational therapy classes and exhibited a keen interest in the musical items presented. He also showed a certain amount of insight into his condition and was ready to discuss his future plans. He was discharged as improved in September, 1953, on a maintenance dose of six tablets of sodium succinate per day. In November he started working on his farm again and soon was seen going out with his friends. He also attended several dances and other social functions. When examined in December he appeared to be quite well; he stated that if he forgot to take his tablets his head "was not so clear". The family reported in January, 1954, that he was fully employed on the farm and maintained his state of social remission.

CASE XVII.—C., aged thirty-eight years, was admitted to hospital in May, 1953, with delusions and ideas of grandeur. He appeared to be well orientated in time and his memory of both past and more recent events appeared to be quite clear; but he had no knowledge of where he was, nor could he explain why he was in hospital. A provisional diagnosis of paranoid schizophrenia was made and electroconvulsive treatment was commenced. A full course of shocks produced no effect. In October he was started on a course of 36 succinate injections in addition to three grammes of sodium succinate given orally, the latter part of his treatment being continued beyond his course of injections. His delusions disappeared entirely during this treatment and his outlook on life became normal. He stated spontaneously that he felt very much better and more cheerful. In December "Pentothal" was administered intravenously in an attempt to elicit whether his previous delusions had fully disappeared or were merely dissimulated; none could be elicited. He was then permitted to go on trial leave. In January, 1954, he returned for a further period of observation; it was found that the improvement had been maintained.

CASE XXVII.—D., an emaciated little man of forty-six years, was admitted to hospital in January, 1953, with a life-long history of maladjustment, commencing with truancy at school and leading through all forms of being a ne'er-do-well, finally to his certification. He had neglected himself, had refused to eat, and was disinterested and apathetic and admitted to auditory hallucinations. In November, 1953, he was given a month's course of barbiturate-succinate injections, during which time his mental condition improved greatly. He became interested in his surroundings, and began to take care both of himself and of his belongings; he began to read newspapers and books, and his auditory hallucinations ceased. His previously depressed outlook on life became more hopeful: "The whole world seems brighter since the treatment started." He also manifested a strong desire to start some useful work. His release on trial leave is being considered.

CASE XXVIII.—Mrs. E., aged thirty-five years, was admitted to hospital in May, 1953, as a mute catatonic, just sitting or lying in bed without any attempt to look after herself; she had to be spoon-fed. Her husband, a clerk—a vague, mild, middle-aged man, possibly helpless, though apparently genuinely concerned—stated that they had had a courtship of five years' duration because he had hesitated to marry, considering it his foremost duty to look after his widowed mother. They finally did marry, and have since lived together with his mother in one big house. According to the records, Mrs. E. fell mentally ill after the birth of her second child in March, 1951, was admitted to hospital and later released as being "fairly well", had relapsed in May, 1951, was again in hospital, and was released in September, 1951, as improved, though she was said to be not quite her former normal self. Electroconvulsive treatment had remained without beneficial effect. In July, 1953, she was given a full course of barbiturate-succinate injections and improved sufficiently to be discharged in September with instructions to continue with the oral ingestion of six tablets of sodium succinate per day. In spite of several letters requesting her return, she did not present herself either for reexamination or for further issues of the tablets. Inquiries made at her home revealed that her relations did not worry about the patient taking the tablets

as long as she remained well. She was transferred to an undisclosed hospital when she relapsed in December, 1953, and she is no longer under our care.

Comments.

Over 50 schizophrenics have now already been treated by the method presented, modified, of course, by the particular needs and response of each patient. Thus, some patients received only six to eight barbiturate coma treatments, and a few had none at all. In other cases, three to six electro-convulsive treatments were used in order to achieve a rapid improvement if possible, which was then maintained by succinate given intravenously or orally. No beneficial effect was observed in hebephrenics and in those schizophrenics whose illness was of long duration (and who were just dull and unreactive deteriorated subjects). All other patients showed some degree of good response; about one-third improved rapidly to such an extent that prolonged leave or even release could be considered; another third showed distinct improvement; whilst the rest improved sufficiently to be at least more easily manageable in the ward. It appeared that prolonged oral succinate medication was essential to maintain the initial improvement. All patients who stopped taking the tablets within the first few months after release relapsed in a few weeks, though they responded again when the treatment was recommenced. On the other hand, none of those who continued the oral treatment as instructed has yet relapsed. The initial improvement was not only maintained, but it was frequently, sometimes stepwise, increased during the following months. It is, of course, too early to decide whether these improvements should be considered as temporary remissions, or whether they promise a more permanent cure. In this context it is noteworthy that the best results were obtained when it was possible to release the patients at an early stage of improvement into more suitable surroundings than the usual hospital ward can provide, so that their final steps of improvement coincided with their adjustment to a more active life. Difficulties were encountered in the cases of several European migrants, because these patients had practically no knowledge of English and thus it became nearly impossible to find suitable homes for them. Although these patients do attend English classes in the meantime and are kept occupied within the hospital premises, it is feared nevertheless that the often unavoidable delay in their release from confinement may interfere with their process of recovery.

We do not believe that our observations so far justify an extensive discussion of the theoretical background of the therapy, which even in the case of insulin or electro-convulsive therapy is still very much in debate. We content ourselves, therefore, with merely mentioning our rationale in broad outlines. Barbiturates in low concentration affect different regions of the brain, the basal ganglia and the hypothalamus, to a different extent (Wyke, 1952; Wilkins *et alii*, 1949), and lower or abolish the functional activities of the nerve cell before the metabolic activities are seriously affected (Brink *et alii*, 1951). It is thus likely that a barbiturate acts on some centres earlier than on others. It may act as a relaxant in so far as it releases attitudes of tension, anxiety *et cetera*, as is shown in everyday clinical use of these drugs; this same mechanism may also release subconscious or suppressed emotions and conflicts and bring them temporarily to the surface. In the normal course of barbiturate narcosis in a schizophrenic all these disturbances rearrange themselves during the period of waking up, and even prolonged barbiturate narcosis alone has no effects other than possibly relieving one temporarily prominent symptom like excessive agitation. Analeptics also may affect different regions of the brain in a different degree, and succinate mainly appears to cause a more intensive and more pleasant awareness (Trautner *et alii*, 1953; Schueler, 1948); "the world looks so much clearer and brighter", as the patients state again and again in relating their experiences after intravenous injection. We believe, therefore, if at an appropriate moment during barbiturate narcosis an analeptic was applied which affected the various centres differently from the barbiturate, that it may modify the sequence of waking up in such a way that some released emotional material may, at least temporarily, remain within the sphere of consciousness and be dealt with at a conscious level. We contend that essentially the patient is helped to deal with his own problems, and that our therapeutic procedure only facilitates his own processes of mental clearance or elucidation. (This consideration, of course, does not exclude the additional effect of psychological assistance from the attending doctors, nurses and others.) We believe that, after a series of combined barbiturate-succinate treatments, mere oral administration of sufficient amounts of sodium succinate may suffice for maintaining and even further improving the patient's awareness and his deepening relationship with his social and professional environment. Our line of reasoning just described is strengthened last, but not least, by the fact that good results were obtained in cases in which withdrawal from the surrounding world of reality into one of spectres was a prominent symptom in the process of disease.

It is always rather difficult to assess the merits of some particular new psychiatric treatment, and considering the limited number of patients treated till now, as well as the limited time of follow-up, we dare not even attempt to do so. But we can justly claim to have brought to a stage at which they could be discharged from hospital, a number of patients who previously had failed to respond to insulin or electroconvulsive treatment, and to have significantly improved the condition of a number of other patients who statistically had a very much lower chance of remission. We think, therefore, that we have made some slight inroad into the vast bulk of refractory cases, and that this fact may justify our continuing with our investigations and even extending them to other sedatives and analeptics which may have still more marked specific effects. It is considered that, even in its present relatively crude form, the method of succinate treatment as a last therapeutic attempt before neurosurgery is entirely worthy of a trial —especially in those cases in which all other non-surgical treatment had previously failed, and in which only neurosurgery remains as a last hope.

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The authors wish to thank Professor R. D. Wright, Department of Physiology, University of Melbourne, Professor F. H. Shaw, Department of Pharmacology, University of Melbourne, and Dr. J. V. Ashburner, Psychiatric Superintendent, Kew Mental Hospital, Melbourne, for their interest in the work outlined, and Dr. Ashburner particularly for his facilitation of the necessary clinical trials.

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Reviews.

Progress in Ophthalmology and Otolaryngology: A Quadrennial Review. Part I—Ophthalmology, edited by Meyer Wiener, M.D., and A. Edward Maumenee, M.D.; Part II—Otolaryngology, edited by Percy E. Ireland, M.D., and Joseph A. Sullivan, M.D.; Volume I; 1952. New York: Grune and Stratton. London: William Heinemann (Medical Books), Limited. 9" x 6¼", pp. 680, with 56 illustrations. Price: £5.

THIS book undoubtedly fills a manifest need, and should be considered a "must" for inclusion in the libraries of ophthalmologists and otolaryngologists who wish to keep themselves up to date, and who have been too busy to cull for themselves all the worthwhile material from the various scientific publications in their respective specialties.

The joint editors are to be congratulated on having assembled such an impressive number as seventy-two American and Canadian contributors, many of whom are recognized international authorities in their respective fields, and chiefs or directors in those medical centres which are so frequently visited by post-graduates from overseas. They include not only ophthalmologists and oto-rhino-laryngologists, but also biophysicists, psychologists, pathologists, radiologists and radiotherapists.

The volume, which is well set out and provided with a full index, is divided into two parts, the first dealing with progress in ophthalmology and the second with progress in otolaryngology, and although it does not purport to be a text-book, there is hardly a branch or aspect in either specialty which has not been considered or dealt with in some manner, no matter how brief. Obviously it has not been practicable in such a work to go into great detail in every subject.

When a contributor has of necessity been obliged to be brief, he has provided readers with an adequate bibliography; such a section is one entitled "Industrial Ophthalmology Since World War II"; it consists of only four pages and is contributed by Kuhn. On the other hand, much more space has been given to the article by Lathrop on the "Surgery of Ménière's Disease"; this is most satisfying and informative. The same statement holds for Sullivan's contribution "Recent Advances in the Surgical Treatment of Facial Paralysis and Bell's Palsy".

The presentation has been surprisingly methodical, each of the two parts being subdivided into four sections.

The ophthalmic part is set out as follows: (*a*) "Basic Science in Ophthalmology" (7 contributors); (*b*) "Diagnosis and Treatment of Diseases of the Eye" (19 contributors); (*c*) "Surgery of the Eye" (5 contributors); (*d*) "Related Subjects in Ophthalmology" (4 contributors).

The ear, nose and throat part is differently subdivided, as follows: (*a*) "The Ear (13 contributors); (*b*) "Nose, Sinuses and Nasopharynx" (8 contributors); (*c*) "Larynx, Tracheo-Bronchial Tree and Esophagus" (4 contributors); (*d*) "Allergy" (3 contributors).

The last-named section includes articles on the "Autonomic Nervous System in Relation to Otolaryngology" and the "Basis for Use of Cortisone and ACTH in Treatment of Oto-rhinologic Conditions", aspects of rhinology and otology which previously have not been thoroughly understood by many ear, nose and throat surgeons, and which are most informative.

Notwithstanding the high standard of this publication, one wonders in these days of extreme specialization why this book in fact exists in its present form. Surely it would be better to divorce the two halves of the book from each other and to produce two different but equally acceptable books, one on ophthalmology and the other on otolaryngology.

A Text-Book of Pathology: An Introduction to Medicine. By William Boyd, M.D., Dipl.Psych., M.R.C.P. (Edin.), F.R.C.P. (Lond.), F.R.C.S. (C.), LL.D. (Sask.), D.Sc. (Man.), M.D (Oslo), F.R.S. (C.); Sixth Edition; 1953. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9¼" x 6", pp. 1024, with many illustrations, some in colour. Price: £6 14s. 6d. from Lea and Febiger.

THE sixth edition of Boyd's "Pathology" is now indeed a monumental work of over one thousand pages and with more than six hundred illustrations. The first edition appeared in 1932 and its popularity has been shown by several reprints of each succeeding edition and by its translation into Portuguese and Spanish. A glance through the contents shows how justified this popularity is, a popularity supported by pathologists, their students and the profession at large. For here we have not only the usual pathological lesions described and discussed and their clinical relationships emphasized, but in smaller print many more rare conditions are noted. The work is thus a storehouse of knowledge to which the bewildered general practitioner can turn when he encounters one of these unusual diseases. Each chapter has at the end a list of works that may be consulted if necessary. There are paragraphs on atomic bomb radiation, heredity in disease, constitution in disease, endocardial fibroelastosis of infants, sudden cardiac death, disseminated *lupus erythematosus*, temporal arteritis, giant-cell pneumonia, beryllium pneumonitis, deficiency hepatitis, the desmoid tumour of the abdominal wall, *papillitis necroticans* of the kidney, various forms of encephalitis (but not Murray Valley encephalitis and its forerunner, Australian X disease), and many other uncommon pathological conditions—even "Dilantin" is mentioned as causing gingival hyperplasia.

Under "Tænia Echinococcus" (page 200), it is stated that human infection is usually due to the eating of unboiled vegetables soiled by the excreta of dogs. In Australia it is due to close association with infested dogs whose surroundings and coats are contaminated from their droppings. Dogs are infected by eating the livers and lungs of cattle and sheep containing hydatid cysts and not by eating the flesh of these animals. The echinococcus cyst shown in Figure 97 is a degenerated cyst with folded membrane. Figure 283, gumma of the liver, looks more like a very degenerated hydatid cyst with pultaceous contents than a gumma, which should show much scarring and contraction round its periphery. The description under "Echinococcus Alveolaris" of exceedingly numerous cysts "varying in size from the microscopic to that of a grain of seed or a pea" suggests the seed hydatid cysts as met with in bone and muscle and not the illusive (to us) alveolar hydatid. The account of *status thymico-lymphaticus* gives a reasonable view of the present situation, that is, one of doubt. Psittacosis is more than a disease of South American parrots—our own parrots and other birds have it in the wild state and the fulmars of the Faroe Islands conveyed the virus to the human inhabitants. The mild rat-derived endemic typhus is not clearly distinguished from the often fatal epidemic form. The thirty-four pages on the pathology of the skin cover the ground well. It is good to see that dental pathology is not overlooked.

Students have always liked Boyd's "Pathology", and so does the general practitioner. With its clinical applications, it is indeed an introduction to medicine—and this edition introduces us to new conditions such as the epidemic hæmorrhagic fever of Korea. If one can say so of such a compendious work, it is (relatively) easy reading, and the text is enlivened by apt quotations and picturesque similes and analogies. It is well worth getting in spite of the price.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Digital Plethysmography: Introducing a Method for Recording Simultaneously the Time Course of the Rate of Blood Flow Into and Out of the Finger Tip", by George E. Burch, M.D., F.A.C.P.; 1954. New York: Grune and Stratton, Incorporated. 8¼" x 5¼", pp. 142, with 83 illustrations. Price: $5.00.

The object of the book is implicit in the subtitle.

"Pediatric Problems in Clinical Practice: Special Medical and Psychological Aspects", edited by H. Michal-Smith, Ph.D., with fourteen contributors; 1954. New York: Grune and Stratton, Incorporated. 8" x 5¼", pp. 320, with four text figures. Price: $5.50.

The book contains thirteen chapters dealing with the problems of children who have recovered from various illnesses.

"Manual of Clinical Mycology", by Norman F. Conant, Ph.D., David Tillerson Smith, M.D., Roger Denio Baker, M.D., Jasper Lamar Callaway, M.D., and Donald Stover Martin, M.D.; Second Edition; 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 8" x 5¼", pp. 468, with 202 illustrations. Price: £3 1s. 9d.

Well illustrated and documented.

The Medical Journal of Australia

SATURDAY, JULY 31, 1954.

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THE EAST WIND.

UNTIL the invention and extended use of aeroplane and glider in the twentieth century the winds were regarded as two-dimensional in direction, and it was natural that the aspects of the human body, front, back, right and left, should have led to the winds being represented as four in number. This concept is abundantly present in the sacred writings of the Hebrews and was carried on into Christian times: Jeremiah XLIX. 36: "I will bring the four winds from the four ends of heaven." Daniel VII. 2: "The four winds of heaven burst forth upon the sea." Revelation VII. 1: "I saw four angels standing at the four corners of the earth, holding the four winds of the earth." The classification of the winds into four types is widespread amongst primitive races as anthropology tells us. In what is perhaps the most poetical portion of Longfellow's Hiawatha the deities directing the winds are four: Mudjekeewis ruled over the west wind, the young and beautiful Wabun had the east wind, the lazy, drowsy Shawondasee controlled the south wind, whilst to the fierce Kabibonokka was given the north wind bringing snow, ice and winter storm. Let it be remembered that the scene of Hiawatha is laid in the lake district of North American where the great land mass is to the west and the sea with the Gulf Stream is to the east, thus reversing the orientation in Europe. It will be remembered that Kipling in his poem "The English Flag" describes the "winds of the world" as four in number, the north, south, east and west. The Hebrew language, not being capable of compound expressions like north-east, used the four names rather freely to denote intermediate directions; according to the apocalyptic Enoch there were twelve winds, and of these four were harmful. Navigation soon made necessary bow, beam and quarter on each side as well as fore and aft, and when the compass card came into use a minuteness of subdivision took place which the landsman to this day rarely employs. It is interesting to note that special names are given to prevailing winds with outstanding characteristics in many parts of the world. The visitor to Cape Town, Morocco or Malta soon learns the terms applied. No one can remain long in Geneva without hearing about and also experiencing the *bise* from the north-east, the *vaudaire* or *Föhn* from the south-east, the *sudois* from the south-west and the *joran* from the north-west. Australia has its brickfielder, southerly buster, cock-eye Bob, Fremantle doctor and the furious willy-willy of the tropics.

In those parts of the world which have contributed most to western civilization the east wind is held in particular dislike. In Syria it may bring sunshine and warmth in winter, but during the rest of the year it can be withering to vegetation and disagreeable to human beings. In the Lebanon district the name for it is *samum*, the same as in Arabia, meaning poison. In this connexion one may note that the Mediterranean *sirocco* derives its name from the Arabic for east wind. In the Old Testament the east wind is mentioned 16 times and always in a derogatory sense; it withers plants, causes springs to dry up, reduces the yield of grain and begets damaging storms. The east wind in Britain is proverbially unpleasant. Mr. Jarndyce in Dickens's "Bleak House" remarks: "I am always conscious of an uncomfortable sensation when the wind is blowing in the east"; Izaak Walton wished his disciple "if he be an honest angler the east wind may never blow when he goes a-fishing". Charles Kingsley in an attempt to put in a good word for the "wild north-easter" stated his view that it produced hardy men. Post-Darwinian biology would explain the fact, presuming it is a fact, by postulating that this bitter and searching wind in winter had eliminated all the weaklings. There is a story of an elderly Englishman who when he saw from a window the weather-cock pointing east, resolved to stay indoors. A waggish boy, hearing about this habit, climbed up and lashed the weather-cock to east and so kept the old gentleman imprisoned in his house for weeks. The story may be *ben trovato*, but could easily be possible.

Why is the east wind so repellent in north-west Europe during the colder months of the year? The answer is really simple and involves some elementary physiology. The epidermis is from the standpoint of physical chemistry a gel containing "water of jellification", as it has been inelegantly named, to the extent of some 30%. The corkscrew course of the sweat ducts as they traverse the external layers of the skin is explained by the fact that one of the main functions of perspiration is to maintain the proper amount of water in the epidermal gel. Now the epidermis when exposed to dry air, particularly when this is in rapid motion, loses much of its water of jellification and this has to be replaced by the sweat. If restitution does not occur fissuring of the skin ensues. If the air is hot as well as dry then the sweat glands in the human being become very active, and weighing of the body demonstrates that though no external moisture is visible the evaporation is extensive. In cold weather it is plainly against the interests of the body as a whole for skin water to be evaporated, considering the high latent heat of evaporation of water; yet evaporation must occur through

physical considerations, and the water content of the skin must be kept up to standard else shrinking and cracking will take place. This is really the secret of the bad name which the east wind has acquired in English literature and ordinary speech. In cold weather the heat loss from epidermal evaporation can submit the temperature-regulating mechanisms to considerable stress, and if through age or debility the metabolism is not correspondingly increased a fall of body temperature is produced, and though a relatively large rise of temperature is tolerated, as in athletic exercises, the slightest threat of a fall is vehemently resisted when possible. Australia fortunately is not afflicted with the British east wind, though in Victoria, south of the Divide, the north wind in winter can be uncomfortable through the factors named. Kingsley's argument that the inclemency of the north-easter produced a hardy type of manhood can logically be applied to any agent which taxes the body's powers; a future and local Kingsley might laud the high humidity, heat and insolation of tropical Australia; but, as stated, the argument is unsound. Climatic conditions uncomplicated by special incidence of disease are rare in most parts of the world and it is here that Australia occupies a very favourable position. It may be truthfully said that nowhere in the world can the physiological effects on the body of climate, including, of course, prevailing winds, be studied with such chance of success.

Current Comment.

FAMILIAL CANCER OF THE COLON.

ALL polypi of the large intestine, according to R. A. Willis,[1] are predisposed to carcinomatous change, the predisposition being least in the solitary polypus and greatest in familial polyposis. He goes on to state that familial polyposis may arise in several successive generations, may be transmitted by either sex and affects males and females about equally. The polypi are not congenital, but usually commence appearing between the ages of fifteen and twenty-five years; so that what is inherited is not polyposis as such, but a special proneness of the colonic mucosa to neoplasia. Familial polyposis almost invariably leads to carcinoma, most of its victims dying of carcinoma of the colon or rectum at an early age, usually between twenty-five and forty-five years. This feature of the disease was well brought out in the melancholy genealogical tables of ten families observed by J. P. Lockhart-Mummery and C. E. Dukes[2] over a period of fourteen years. Lockhart-Mummery and Dukes used the term "familial adenomatosis" for the disease; but recently M. W. Durham[3] has suggested that in view of the fact that malignant change occurs in almost all, if not all, cases and to prevent the engendering of a false sense of security by calling the condition polyposis, familial cancer of the colon would seem a more apt term. Durham presents the history of a family in which 18 out of 29 members traced have familial polyposis or carcinoma of the colon.

In recent years, Durham states, treatment of this somewhat discouraging disease has become more or less standardized and now includes both the prophylactic removal of all cancerogenic tissue, either by operation or by fulguration, and also the radical excision of the bowel after cancer has developed. For the prophylactic treatment, ileo-sigmoidostomy is preferred, followed by total colectomy and repeated fulguration of the polypi in the rectum and lower part of the sigmoid colon. It is agreed that there is a slight chance of cancer developing even in the rectal stump of patients under such management; but with patients who would ill adapt themselves to a permanent ileostomy, Durham considers that there is no doubt about whether the short rectal stump should or should not be retained. Once cancer of the bowel has developed, its radical removal together with the affected proximal part of the bowel will usually entail a combined abdomino-perineal resection with colectomy and permanent ileostomy, since, in the majority of cases, the cancer appears in the rectum. The number of stages to complete the colectomy are now kept to a minimum because of the disadvantages associated with multiple procedures. Among these disadvantages Durham includes prolonged stay in hospital, increased expense, refusal of some patients to undergo subsequent stages, technical difficulties encountered in the later stages, and the effect on the morale of the other members of the family, who probably will face the same procedure.

[1] "Pathology of Tumours", 1948.
[2] *Lancet*, September 9, 1939.
[3] *West. J. Surg.*, January, 1954.

SURGICAL TREATMENT OF AORTIC ANEURYSMS.

A REPORT from Henry T. Bahnson[1] suggests that excision and replacement with a homograft may prove to be the most successful method of treating aortic aneurysms. He points out that the higher mortality of patients with abdominal aortic aneurysms is due chiefly to rupture of the aneurysm. He quotes J. E. Estes, junior (1950),[2] who in a study of 102 patients with such aneurysms found that 33% had died in one year and 90% were dead at the end of eight years, whereas in a comparable age group of the general population only 35% had died in eight years.

To overcome the danger of rupture attempts have been made to strengthen the aortic wall by inducing fibrosis with foreign material wrapped around the vessel, and by coagulation of blood on the weakened wall by means of wires inserted into the lumen. The author does not think that either of these methods is satisfactory; nor is ligation, for even when it is done slowly or in stages ischæmia of the legs often results. Diagnosis of an abdominal aortic aneurysm usually presents no difficulty. Aortography, though helpful in giving some idea of the extent of the aneurysm, is not essential and was not done in all Bahnson's cases. Pain due to the aneurysm which requires narcotics for relief is, Bahnson suggests, one of the indications for operation. The younger the patient, the greater the chance that he will die from rupture of the aneurysm, and hence will be likely to benefit from the operation; conversely it is hardly worth while subjecting to such a major procedure patients who are likely to die in a short time in any case from other causes. Two patients, one of whom survived, were operated on after the aneurysm had ruptured.

The operation is performed through a left paramedian incision, and when the extent of the aneurysm is determined an appropriate homograft can be selected. Homografts are preserved in a special solution at 4° C., or else dried and frozen. Bahnson prefers the latter method, as it allows grafts to be kept almost indefinitely. In general only those aneurysms beginning below the origin of the renal arteries are suitable for operation. Segments of the aorta from just distal to the renal arteries down, to include the proximal part of the common iliac arteries, have been successfully resected and replaced by a homograft. One patient in whom the renal arteries were occluded for 110 minutes during the operation died subsequently from uræmia. In this patient the aneurysm had ruptured and the operation was done as an emergency.

After the operation three patients required amputation for ischæmic changes in the legs. In one patient bilateral amputations were performed; but even so, according to the author, the patient remained grateful for the relief of

[1] *Circulation*, April, 1954.
[2] *Circulation* (1950), 2: 258.

pain previously present in his aneurysm. Thrombosis of the iliac arteries just distal to the clamp with subsequent embolism was held responsible for ischæmia of the legs. This complication has been avoided in subsequent cases, in which heparin, 10 milligrammes, was instilled into the iliac arteries as soon as these vessels were occluded. Besides the patient who developed uræmia two others have died—one in four months from progressive myocardial failure, due to coronary sclerosis, and the other in five months from myocardial infarction. The eleven surviving patients have remained well during periods of observation from two to eight months. Though further work will be required to assess the value of this operation, these preliminary results are encouraging.

TEMPERATURE, PULSE RATE AND ROUTINES OF LIVING.

IT is well known that the temperature of man has a typical daily variation. According to B. A. Houssay's "Human Physiology", the maximum temperature occurs between 5 and 8 p.m. and the minimum between 3 and 5 a.m., the temperature curve being related to the variations in muscular activity and metabolism which are at their maximum in the evening and at their minimum in the early morning. It is further stated that an inversion of the usual cycle is experienced in some instances, for example, in some night workers or in subjects travelling around the world who continue with their usual rhythm, although the day in the place from which they started corresponds to the night in the Antipodes. Nathanial Kleitman,[1] in a general review of the subject, states that this diurnal body temperature rhythm in man is developed by acculturation and maintained by and through adherence to the socially prevalent routine of living. At the same time, there are distinct individual variations in mean composite diurnal temperature curves which have an interesting bearing on the efficiency of the individual. So-called "morning types" reach their maximum temperature early in the day. Their performance is better immediately on getting up in the morning than just prior to going to bed at night. At either end of the waking period, however, temperatures are rather low and performance is poor. A morning individual may, by the end of two or three hours after he gets out of bed, have reached his body temperature peak and his performance will then begin to show a downward trend. Conversely, an "evening type", who comes to life towards the end of the waking period, dislikes going to bed at the conventional retiring hours and hates to get up in the morning. He will show a rise in body temperature curve during the day, and his performance will fit into a corresponding curve. The "in-between individual", whose body temperature rises till the middle of the afternoon and then begins to fall, will show an increase in efficiency followed by a gradual decrease. There are many gradations between the two extremes, and one notably different temperature curve found in a few individuals consists of an early morning rise followed by a temperature plateau for the rest of the day and sometimes well into the evening.

On the question of the shifting or reversal of the diurnal body temperature curve by changes in daily routine, Kleitman quotes the case of a twenty-seven-year-old bedridden idiot whose body temperature curve was reversed by changing the hours of illumination and feeding. Attempts to establish body temperature rhythms of more or less than twenty-four hours by changed routines of living have led to success in some cases and failure in others. The length of the artificial period is said to be of importance. Adjustments can be made to periods of twenty-one hours and twenty-eight hours, but not to periods of forty-eight or twelve hours. Further light is thrown on this aspect of the subject in a recent paper by Kleitman and Esther Kleitman,[2] in which they describe the results of attempted abolition of twenty-four-hour rhythm under summer conditions in the Arctic region where the sun does not set. Four members of a family, consisting of two middle-aged parents and two young adult daughters, had lived on an artificial, twenty-one-hour or twenty-eight-hour sleep-meal-activity schedule in two localities in the United States. This had led to the gradual development of distinct body temperature curves corresponding to the new periodicities in some subjects, but the male parent did not deviate from the usual twenty-four-hour rhythm. It was thought that if the family lived together in the continuous daylight of the Arctic summer, conditions would be more favourable for the abolition of the twenty-four-hour rhythm; so they spent nine weeks, from May 21 to July 23, 1951, at Tromsö, Norway, which is located well above the Arctic circle. Two three-week periods of artificial living routines, one of eighteen hours (including six hours of sleep) and the other of twenty-eight hours (including eight or nine hours of sleep), were separated by two weeks on the usual twenty-four-hour schedule. Owing to illness, the data relating to the female parent could not be used. The usual diurnal oral temperature rhythm of the two young women was disrupted, and they were able to make at least a partial adjustment to the artificial sleep-wakefulness schedule; but the male parent proved resistant, as he had been under the conditions in the United States, to an alteration in oral temperature rhythm. His subjective experiences and other data relating to his activity corresponded to this failure of adjustment. He had great difficulty in sleeping during the daytime hours, and when sleep did supervene it was much less restful than at night.

One of the particular objects of this investigation was to study the relation between heart rate and body temperature in man. In general terms, it has long been recognized that there is a relation between heart rate and body temperature, and it is said that, before the introduction of the thermometer, physicians often judged the degree of fever of a patient by the rapidity of the pulse. Moreover, it has been the usual observation that alterations in the diurnal body temperature curve have been accompanied by a similar change in the heart rate. However, despite the male parent's resistance to alteration in his temperature rhythm, his heart rate quickly became adjusted to both the eighteen-hour and the twenty-eight-hour schedule of rest, activity and food intake, and it is clear that rest in bed, whether or not it results in sleep, serves to produce a slowing of the heart rate. The Kleitmans suggest that this particular adjustment of heart rate may be due to the capacity of the subject concerned for muscular relaxation; but they draw the reasonable general conclusion that the diurnal heart rate is not what Kleitman has called elsewhere a physiological rhythm, like the diurnal temperature curve, but a simple periodicity, which is immediately coupled with the daily routine of living.

One point of interest emerging from this study is the fact that adaptation appears to be more rapidly made to an artificial cycle longer than twenty-four hours rather than to a shorter period. This fact can be borne in mind if for any reason a departure from the normal diurnal routine of living is at any time necessary. As possible circumstances requiring such an adjustment, the Kleitmans instance the introduction of an atomic power plant which will permit a submarine to cruise for weeks without surfacing, and (unhappy thought) a demand from the exigencies of the atomic age for the establishment of subterranean communities.

[1] *Phys. Rev.*, January, 1949.
[2] *J. Appl. Physiol.*, November, 1953.

THYMECTOMY AND MYASTHENIA GRAVIS.

SINCE 1936, when the American surgeon A. J. Blalock successfully removed a thymoma with alleviation of associated *myasthenia gravis*, the role of thymectomy in the treatment of *myasthenia gravis* has been a subject of dispute. Blalock followed up his initial success by removing the apparently normal thymus gland from six patients suffering from *myasthenia gravis* with favourable results. This work came to the notice of Geoffrey Keynes, in London, and since 1942 he has performed the operation

in a large number of cases with satisfactory over-all results. These historical details and a thoughtful discussion of many aspects of surgery of the thymus gland are set out in an oration delivered by Keynes before the Medical Society of London in May of this year and recently published.[1] The whole oration will be of interest to many, but attention should be drawn to the light thrown by it on the present status of thymectomy in relation to myasthenia. When the subject was discussed in these columns last year,[2] mention was made of the adverse criticism of the procedure emanating from L. M. Eaton and O. Clagett, of the Mayo Clinic. As it happened this criticism was referred to only in passing, and no inclination was shown to join the ranks of "writers of leading articles" who are described by Keynes as trying "to discredit the surgical treatment of myasthenia gravis with douches of cold water drawn from the Mayo Clinic cistern". It is interesting to note now that Eaton and Clagett have reversed their previously held opinions. Keynes refers to a contribution by Eaton and Clagett, together with J. A. Bastron, presented at a meeting of the Association for Research in Nervous and Mental Diseases, held in New York, in December, 1952, in which they set out the evidence for a relationship between the thymus and *myasthenia gravis* and reexamined the place of thymectomy in its treatment. A strong criticism made by Keynes of their previous approach to the subject is that they did not separate their tumour patients from their non-tumour patients, failing to appreciate what Keynes regards as an essential point—namely, that the disease in these two kinds of patients runs a different course and demands a different therapeutic approach. In their 1952 paper they state that from the data presented "it is readily seen that patients who have thymomas do not get along as well as those who do not have thymomas". Having accepted the need to consider these two classes of patients separately, they conclude that "in a comparison of surgical and non-surgical patients who did not have thymomas the better results are found in the surgical group". They continue: "In this latter group 45·8 per cent. of the patients obtained a result justifying the risk, discomfort and expense of surgical treatment; this is contrasted to an incidence of such results in only 16·2 per cent. of the patients in the non-surgical group." Keynes is quite unable to resist the temptation to say, in effect, "I told you so", and his criticism of Eaton and Clagett is trenchant. Unfortunately, their paper is published in a highly specialized neurological volume with a limited circulation, and Keynes is concerned at the fact that while the previous criticism has had wide publicity, the recent *volte-face* may not so readily become known. However, he has done what he can to remedy the lack of publicity, and we may regard the place of thymectomy in the treatment of *myasthenia gravis* as being now well established. It is pleasing to note that Keynes concludes his account of the controversy on a philosophical note. The whole affair is, as he remarks, an interesting study in the elusive subject of the formation of public opinion. He refers to "the cool way in which we, as a profession, regard our novelties", looking at them carefully all round before we admit them to the canon of accepted practice; and he concludes: "This deliberation may be irksome to the individual because life is short, but it is perhaps wholesome in the long run."

CARCINOMA-IN-SITU OF THE LARYNX.

THE term carcinoma-in-situ was used by Broders in 1932 to describe an early pre-invasive stage of intraepithelial carcinoma that is recognized by the profound cellular alteration in intact surface epithelium. According to A. H. Miller and H. R. Fisher,[3] usually the layer of epithelium involved is increased in thickness, but this is not invariably so, as the same microscopic changes can be seen in extremely thin epithelial surface layers. There is always a clear-cut, regular and continuous demarcation between epithelium and connective tissue. Miller and Fisher record details of 13 cases of carcinoma-in-situ encountered in five years in the department of oto-laryngology of the University of Southern California. The average age of patients was forty-six years. Ten were males, and three were females. All suffered from hoarseness. Seven were heavy smokers, four smoked moderately and two did not smoke at all. A clinical diagnosis was not made in any case prior to biopsy, and the appearance of the lesion in only two cases suggested that there might be malignant change. Leucoplakia was prominent in eight cases; in others there were smooth, red swellings. In seven cases all of one cord from anterior to posterior commissure was involved, so that a laryngo-fissure operation could not be expected to permit safe removal of all diseased tissue. Ten of the patients were treated by fractional deep X radiation, the superficial lesion being first stripped away in seven cases. In seven of the ten cases in which treatment was by irradiation, the condition appears to be clinically controlled after intervals of six months to four and a half years. In two others a recurrence of carcinoma-in-situ has appeared six months and sixteen months respectively after treatment. Two patients were treated through a laryngeal fissure, and the condition is apparently controlled. Stripping was carried out in only one case, and the larynx appears to have remained normal. It is apparent that a fairly wide range of treatment has been adopted in this series, but so far no firm conclusions can be drawn concerning the ideal treatment that should be used.

A REPORT ON ALCOHOL.

THE World Health Organization has committees which deal with alcohol. One is the Expert Committee on Drugs Liable to Produce Addiction, another is the Alcoholism Sub-Committee of the Expert Committee on Mental Health. The latter is concerned with the psychiatric, clinical and social aspects of the question and the former with the physiological, pharmacological and biological properties of alcohol. The Expert Committee on Alcohol has issued its first report. It is Report No. 84 of the Technical Report Series of the World Health Organization. The date of publication is March, 1954.

The first subject discussed is tolerance to alcohol. The Committee recognized that there was a state of confusion arising from the indiscriminate use of the term "tolerance". It is necessary to make a distinction between the use of the term "tolerance" in connexion with addiction-producing drugs of the morphine type and that applicable to the use of alcoholic beverages. In view of the social use of alcohol, it was agreed to regard tolerance towards alcohol as the capacity of the organism to function with alcohol in the blood without a measurable deterioration in nervous function. Experiments have shown that there is a critical level or threshold of alcohol concentration in the blood at which performance begins to show deterioration. This threshold is characteristic of the individual tested and of the particular test employed. Tolerance to alcohol can thus be measured by that concentration of alcohol in the blood at which a demonstrable effect on the performance of a given test of nervous function first becomes apparent. This is a precise method of measuring tolerance to alcohol. It was therefore considered that the term "tolerance" should not be used in other senses, as, for example, the following: (*a*) the amount of alcohol required by an individual to produce euphoria; (*b*) the amount of alcoholic beverages ingested by an individual without the appearance of signs of intoxication; (*c*) the mere ability to ingest large quantities of alcohol irrespective of whether such quantities result in intoxication or not. The Committee states that the main reason for the rejection of these forms of usage was because they relate the effects of alcohol to dosage. This is undesirable, because variation in the rate of absorption and other aspects of the metabolism of alcohol, as well as the period in which ingestion takes place, may change the

[1] *Lancet*, June 12, 1954.
[2] M. J. AUSTRALIA, October 31, 1953.
[3] *Ann. Otol., Rhinol. & Laryngol.*, June, 1953.

relation between dosage and blood alcohol levels. It has been shown that to produce the same effect, as measured by objective tests, a higher blood alcohol level is required in habitual heavy drinkers than in moderate drinkers and abstainers. The acquisition of this increase in tolerance is shown by its disappearance after a period of abstinence. Prolonged heavy intake of alcohol does not increase the rate of absorption, nor does it increase the rate of oxidation or excretion of alcohol. There is no evidence to show that the permeability of the hæmatoencephalic barrier to alcohol is lowered. Two other explanations have been suggested. One is that conditions may arise which make the nervous system more resistant to alcohol; the other that the individual may compensate for the effects of intoxication through the process of learning. It is stated that these two possibilities are often regarded as though mutually exclusive, but it is not thought necessary to make this assumption. The Committee thinks that although the biological mechanism of the change is not fully understood, the total phenomenon cannot be accounted for solely by compensation based on learning; in addition, there is necessary for its explanation a changed physiological response of the organism to alcohol. Whether withdrawal symptoms occur in alcoholism can be discussed only on the basis of clinical observation. Upon interruption of an acute alcoholic episode, certain symptoms are often observed. The symptoms are largely in the category of psychomotor excitement, but convulsions of the *grand mal* type are also seen.

The Committee believes that a sharp distinction should be made between these symptoms and those which are produced by the withdrawal of the morphine type of drugs. The symptoms which occur after sudden withdrawal of alcohol are of short duration, and in the absence of serious organic disease, do not usually have grave consequences. These symptoms are tentatively regarded as involving a process of physiological readaptation. The symptoms occur only on abrupt withdrawal and are not observed either during voluntary or during enforced abstinence from alcohol. This distinguishes the effects of withdrawal of alcohol from those following withdrawal of drugs of the morphine type. For this reason the Committee thinks that in connexion with alcohol, the term "withdrawal symptoms" should be used alone; the term "abstinence syndrome" is justified in connexion with drug addiction of the morphine type.

The approach to alcoholism by animal experiments is discussed. The Committee refers to investigations carried out with rats on the possibility that nutritional deficiencies may be responsible, and it concludes that the point is far from settled. Approach by means of endocrine studies is mentioned, and it is stated that craving for alcohol has been interpreted as arising from the need for substances of the adrenal cortex which, according to the results of certain experiments carried out on animals, may be released after the ingestion of alcohol. It has been surmised, although not yet demonstrated, that alcoholism might result from a disturbance in the control of the pituitary gland affecting adrenocortical secretions. The Committee thinks that the evidence in the literature on this point is conflicting. Several other concepts are referred to, but these need not be set out. The Committee is not convinced by the evidence put forward in favour of any of them. It is pointed out that empirical methods remain dominant in the therapeutic field. It is suggested that a tentative approach is required, which visualizes an interplay of laboratory investigation and planned clinical experiments designed to check and guide the laboratory work. Although there is at present no evidence for the initiation of heavy drinking through organic disturbances, it is thought that this possibility can by no means be excluded.

The question of the position of alcohol as a drug is discussed. The definition of an addiction-producing drug given by the Expert Committee on Drugs Liable to Produce Addiction was accepted. According to this definition, drug addiction is characterized by: (*a*) an overpowering desire or need (compulsion) to continue taking the drug and to obtain it by any means; (*b*) a tendency to increase the dose; (*c*) a psychic (psychological) and sometimes a physical dependence on the effects of the drug. Overpowering desire to continue taking the drug and to obtain it by any means was considered by the Committee to occur only in the case of a minority of users of alcohol. If it does occur, it is thought to be of a lesser degree than is the case with morphine. It was agreed that alcohol could not be classified as an addiction-producing drug. The question was discussed whether alcohol should be listed among the habit-forming drugs and the Committee decided that this was not possible in view of certain of its characteristics. Emphasis was laid on the following points. (i) The serious social consequences of excessive intake of alcohol must exclude alcohol from the habit-forming group of drugs. (ii) The sudden withdrawal of alcohol from heavy drinkers often causes withdrawal symptoms of short duration which imply some degree of dependence, although withdrawal of alcohol does not cause persistent abstinence symptoms as in the withdrawal of morphine and allied drugs. (iii) Although the Committee hesitated to acknowledge the increased voluntary intake of alcohol observed in animals under certain experimental conditions as "experimental alcoholism", these observations seemed to indicate that a "craving" for alcohol may have a physical basis besides the psychological origin which presumably is predominant in alcoholism. Alcohol was placed in a category of its own, intermediate between addiction-producing and habit-forming drugs. The following is the statement finally agreed upon:

> Alcohol must be considered a drug whose pharmacological action is intermediate in kind and degree between addiction-producing and habit-forming drugs, so that compulsive craving and dependence can develop in those individuals whose make-up leads them to seek and find an escape in alcohol. With this substance the personal make-up is the determining factor, but the pharmacological action plays a significant role. Damage to the individual may develop, but does so in only a minority of users. The social damage that arises extends, however, beyond these individuals themselves.

The other matters considered in this report are the metabolism of alcohol and alcohol in relation to traffic. The final statement is that further exploration of the matter would necessitate a joint meeting of experimental workers and clinicians, and the Committee recommends that the World Health Organization should consider the convening of such a meeting.

THE SAME OLD STORY.

THE 1954 edition of the Medical Directory of Australia is about to be set in type, and it is hoped that the volume will be completed before the end of the year. Cards have been sent to all those whose names appeared in the last edition, and also to those who have graduated since that time and whose addresses are available. The response has been typical of most appeals to members of the profession for information or for an opinion. About 40% of persons who were circularized have replied. Some members of the profession will declare later on that they have never received a card. From the office of the Directory it can only be declared that cards have been sent. In our appeal on this subject three years ago, we quoted a passage from a volume printed in 1771, entitled "A Life of Christ", the author being the Reverend John Fleetwood, D.D. At the back of the book there was a list of subscribers, and the following footnote appeared:

> The publifher of this work hopes that no Subfcriber will take it amifs, if he finds his name omitted in this lift, as he has not been able to procure near one half of them, notwithftanding he gave a printed notice for that purpofe, and delayed the publication feveral weeks entirely on that account. It is alfo hoped that no gentleman will be angry if he finds his name fpelt wrong, as they are all printed exactly as they were delivered by the hawkers and others.

Members of the medical profession are warned that the onus of the correctness of an entry is on the individual and not on the publishers.

Abstracts from Medical Literature.

SURGERY.

Hiatus Hernia of the Diaphragm.

C. R. LAM AND L. J. KENNEY (*J. Thoracic Surg.*, January, 1954) review the modern literature relating to the surgical treatment of hiatus hernia of the diaphragm and report their own experience based on anatomical studies of the diaphragm. They believe that the key to successful surgery is the obliteration of an anatomically weak area immediately posterior to the œsophagus, which they call the crural membrane. They describe experiments showing that interruption of the phrenic nerve does not affect the diaphragm in the vicinity of the hiatus and believe that this operation is useless and possibly harmful in the treatment of hiatus hernia.

Cardiopexy in the Treatment of Cardiac Ischæmia.

S. A. THOMPSON AND A. PLACHTA (*J. Thoracic Surg.*, January, 1954) review their fourteen years' experience of the operation of cardiopexy (the introduction of magnesium silicate powder into the pericardial sac) in the treatment of myocardial ischæmia. Fifty-seven patients were treated, of whom 20 were much improved and 25 moderately improved. Five patients received less than 50% improvement. There were seven deaths attributable to the operation, and the average post-operative survival time of 17 patients who have since died is five years. The operation produces first pronounced hyperæmia of the myocardium and the formation of new blood vessels within it; then a foreign-body granuloma is produced which involves the pericardium and epicardium; then adhesive granulomatous pericarditis without constriction takes place, and the myocardial blood supply is further enhanced by the anastomosis of extracardiac with myocardial blood vessels through the granulomatous bridge. This acute inflammatory reaction terminates in chronic adhesive granulomatous pericarditis with extracardiac and myocardial vascular anastomoses, which have been observed to persist for ten years. The authors believe that the chronic lesion remains active for as long as the subject lives. Since the particles of magnesium silicate powder are not absorbed and very little is removed by the lymphatics or phagocytes, the greater part of the powder remains fixed in the adherent tissues of the pericardium and myocardium and serves as a constant and continuous source of irritation and hyperæmia and a stimulus to the formation of new blood vessels. Scar tissue does not form.

Hypotensive Anæsthesia for Radical Surgery.

C. P. BOYAN (*Arch. Surg.*, December, 1953) reports experiences with hypotensive anæsthesia in selected cases of radical pelvic and abdominal surgery at the Memorial Cancer Center, New York. The physiological basis for its use is discussed. The author states that after any pre-operative deficit of the blood volume has been corrected the patient is anæsthetized by intravenous injection of thiopental sodium and tubocurarine chloride, and the peripheral resistance is lowered by the intravenous administration of hexamethonium bromide. This blocks the autonomic ganglia and thus reduces the peripheral resistance in the arterioles. If such hypotension of not less than 75 millimetres systolic pressure is maintained for only sixty to ninety minutes in a patient in the head-down position and with an adequate airway, there will be no changes in the electrocardiogram and no ischæmia of the brain. The results were found to be less satisfactory in young adults because of difficulties in lowering the blood pressure, while the best results were obtained in the group aged forty-five to sixty years. It is stressed that hypotensive anæsthesia should be employed only by competent and very experienced anæsthetists. In a series of 114 cases it was found that the principal blood vessels were more easily and cleanly visualized for isolation and dissection, and areolar tissues and lymph nodes were dissected *en bloc* in a more satisfactory manner than under other methods of anæsthesia with the blood pressure at usual levels. In one case death was probably due to bleeding at the operation site, and in another death was due to a sudden cardiovascular collapse. It was considered that these deaths might have been due to the type of anæsthesia used. At the end of the operative dissection and before the abdomen was closed, the legs were routinely elevated and "Neo-Synephrine" was given by intravenous drip administration when required. Blood lost during or after the operation was immediately replaced, and the systolic blood pressure was restored to at least 100 millimetres of mercury before the abdomen was closed, lest occult bleeding should subsequently occur from an unligated vessel.

Surgery in the Aged.

S. E. ZIFFREN AND S. C. CULLEN (*J. Am. Geriatrics Soc.*, January, 1954) review the principles in the management of the aged patient who requires non-emergency surgery. They state that in some no extensive preparation is needed, but in others anæmia, dehydration, decreased blood volume and deficiency of electrolytes, protein, and vitamins all require correction. The deficient blood volume in aged patients with a serious surgical disease is difficult to estimate and may exist with a normal blood count or plasma protein level. Blood transfusions will help to overcome this deficiency, and then with improved intake of fluids by mouth subsequent blood examination may reveal anæmia. Fluid replacement in the elderly is based on a lower insensible daily loss than in younger patients; and, in general, the fluid used should contain less salt. It is imperative that the blood volume should be replenished before introducing any appreciable amount of saline solution. Even the presence of œdema is consistent with the necessity for rehydration in some patients with cardiac failure; and the correction of the latter will relieve the œdema. Even though malnutrition exists the intravenous administration of protein hydrolysate and glucose can do little to correct it, and by far the most suitable and effective means of overcoming malnutrition is by the oral route; alcoholic stimulants, supplementary feedings, formula feedings and the injection of a rich formula through an indwelling gastric tube—all should be used on occasions. After operation even more care is necessary to avoid the administration of unnecessary quantities of fluids and salts. The main object of the early convalescent period is to tide the patient over until such time as he can take fluids and foods orally. This early period is not the time to attempt overzealous infusions in order to build up an impaired nutritional state.

Trauma to Major Arteries in the Course of Cancer Surgery.

J. W. LORD, JUNIOR, AND C. F. SCHETLIN (*Am. J. Surg.*, March, 1954) state that as a result of several advances in the field of vascular surgery during the past ten or twelve years, the surgeon interested in the eradication of cancer by operative means no longer has to regard the resection of a major artery as a contraindication to radical excision of a lesion. In most of the cases in which a segment of an artery is resected with a malignant neoplasm, an autogenous venous graft is the one of choice unless the aorta is involved, when a homologous arterial graft should be employed. Occasionally, direct end-to-end anastomosis of the artery is possible. In some cases, for example, when there is involvement of the first part of the subclavian artery, simple ligature is the method of choice. Five illustrative cases are described in which these various methods of arterial repair were required; and, because of the continued extension of operative procedures aimed at the eradication of cancer, it is agreed that some training and experience in vascular surgery should accompany general surgical training.

Transdiaphragmatic Approach to the Heart during Cardiac Arrest.

J. W. DEVINE AND J. W. DEVINE, JUNIOR (*Am. J. Surg.*, March, 1954), describe a transdiaphragmatic incision for exposing the heart during cardiac arrest. This approach is advised when cardiac arrest occurs during an abdominal operation or when an adequate respiratory system cannot be maintained with an open chest, owing to lack of equipment or skilled anæsthetists. The advantages are the fact that the heart may be exposed without entering either pleural space, and the ease of occluding the abdominal aorta below the origin of the renal vessels during the cardiac massage. The chief disadvantages are the difficulties of determining whether the heart is in simple arrest or in ventricular fibrillation and of employing the methods to defibrillate the heart when these are required. The abdominal incision is extended upwards above the xiphisternum, and the left coronary ligament of the liver is divided. The diaphragm is then divided between clamps just to the left of the œsophageal hiatus, and the pericardium entered.

Desmoid Tumours.

J. E. STRODE (*Ann. Surg.*, March, 1954) describes the clinical characteristics of desmoid tumours and emphasizes the fact that the surgeon should not be conservative in his treatment. The large defects thus produced may necessitate resorting to the use of

dermal grafts, tantalum, stainless steel gauze or fascia-lata grafts. Three cases of desmoid tumours are described which were seen over a period of twenty-six years. In one case the tumour was first detected when the patient was aged one year and continued to grow till the time of puberty despite irradiation and two attempts at ablation. After puberty some apparently spontaneous regression occurred. In another case a large piece of *fascia lata* was used to cover the defect in the abdominal wall remaining after excision of the tumour. This defect was 14 centimetres in diameter. In the third case almost the entire left half of the abdominal wall was removed together with an adherent loop of sigmoid colon. The defect in the abdominal musculature was closed with two large pieces of *fascia lata*, one from each thigh.

Carcinoma of the Pancreas.

W. B. HUTCHINSON (*Arch. Surg.*, January, 1954) discusses the present status of the operative treatment of carcinoma of the pancreas and concludes that every case requires a careful appraisal based on the patient's general condition and the anatomical site and local behaviour of the tumour. All reported cases show that for practical purposes the natural history of carcinoma of the pancreas has not been altered by radical pancreaticoduodenal resection. This appears to be due to the early stage at which metastases occur. The outlook for carcinomata of the lower end of the common bile duct, ampulla of Vater and duodenum is much more favourable, and in the absence of local infiltration or distant metastases every effort should be made to extirpate them. For the patients with carcinoma of the pancreas partial pancreatico-duodenectomy is justified only if the malignant condition is apparently localized to the pancreatic head. In other cases the palliative procedure will be determined by the local findings, and the one adopted should be simple and involve a minimal number of anastomoses. Of these, cholecyst-jejunostomy, with a comfortable length of jejunum, is the procedure of choice. In a series of 60 pathologically proven cases for which operation was performed, a by-pass procedure to overcome the biliary obstruction was employed in 17 and pancreatic resection in 16. In only three of the latter was total pancreatectomy performed.

Clinical Use of Lævo-Arterenol during Cyclopropane Anæsthesia.

SYLVAN N. SURKS AND NORTON M. LUGER (*Surgery*, January, 1954) state that cardiac arrhythmias occur not infrequently with all general anæsthetic agents. As these effects are more frequently noted in connexion with the use of cyclopropane anæsthesia, most anæsthetists prohibit the use of adrenaline during the administration of cyclopropane. Because of the structural similarity between adrenaline and lævo-arterenol (*l*-noradrenaline), this interdiction has also been transferred to the latter drug. The authors anæsthetized with cyclopropane 45 patients who were at the time receiving lævo-arterenol for the treatment of shock. In none was there clinical evidence of toxicity. The nature of the operative procedures is stated, and it is pointed out that seven of the patients, who were in a state of shock pre-operatively, received lævo-arterenol prior to and during the induction of cyclopropane anæsthesia. Thirty-eight other patients went into a state of shock while undergoing operation, and lævo-arterenol therapy was initiated during the maintenance of cyclopropane anæsthesia. The drug was administered by constant intravenous infusion, the rate (usually one to three millilitres per minute) depending on the adequacy of the restoration of blood pressure. The concentration of the drug used was usually eight microgrammes per millilitre. However, on rare occasions, concentrations as high as 16 microgrammes per millilitre were used. The authors state that while no untoward cardiocirculatory effects from the combinations of cyclopropane anæsthesia and lævo-arterenol were noted, the technique should be employed cautiously and discontinued if cardiac irregularities develop. The simultaneous administration of light cyclopropane anæsthesia and a continuous intravenous infusion of dilute lævo-arterenol does not appear to be as dangerous as has been hitherto believed.

Polyvinyl Sponge in Repair of Abdominal Wall Defects.

T. L. SCHOFIELD, G. A. HALLENBECK, J. H. GRINDLAY AND E. J. BLADES (*Arch. Surg.*, February, 1954) report the experimental use of polyvinyl sponge to bridge defects produced in the abdominal musculature of dogs and rabbits. They state that polyvinyl sponge differs from tantalum and stainless steel gauze in that it is cheap and very easy to handle. It also has the advantages that it is inert, can be sterilized by boiling, is smooth and elastic, and is radiotranslucent. After the excision in seven dogs of portions of the rectus muscle and its sheath measuring eight by ten centimetres, the gap was bridged with a single layer of polyvinyl sponge three millimetres in thickness; in six no herniation occurred. The interstices of the sponge became filled with fibrous tissue, which was continuous at its edges with the scar in the muscle. In the remaining dog, however, infection occurred, and the sponge was not invaded by fibrous tissue; nor did it remain in position. In none of the six dogs in which a double layer of polyvinyl sponge was used to fill in the gap did a hernia develop. Other experiments are described in which was tested the tensile strength of the abdominal wall of rabbits containing a gap bridged with polyvinyl sponge. In these animals it was found that there was a gain in tensile strength for approximately the first forty days, but thereafter there was little or no gain.

Intractable Ulcerative Colitis.

H. E. BACON AND H. D. TRIMPI (*West. J. Surg.*, December, 1953) review their experience with the surgical treatment of chronic ulcerative colitis over the past thirteen years. Of a series of 331 cases operation was performed in 81, with five deaths. The procedures employed included ileostomy by itself or with colectomy or with colectomy *plus* excision of the rectum, ileosigmoidostomy and colectomy, and abdomino-perineal excision. The authors conclude that the operation of synchronous ileostomy and colectomy has proved to be of noteworthy importance; it has all but replaced ileostomy and second-stage colectomy in their armamentarium, since it means removing the large areas of purulent, necrotized bowel at an early stage in surgical management instead of prolonging invalidism between ileostomy and colectomy. Recovery is prompt, the surgeon is not faced with the difficulty of excising a diseased caecum and ascending colon from the vicinity of the ileostomy, and the adhesions are less than after a preliminary ileostomy. It has been found that the complications following ileostomy *plus* colectomy are less than after ileostomy when the diseased bowel is left *in situ*. In most cases the rectum is removed later; otherwise it acts as a focus of infection, and carcinoma may develop in this area despite extirpation of the diseased bowel proximal to it.

Gastro-Ileostomy.

A. D. MCKENZIE AND H. R. ROBERTSON (*Ann. Surg.*, December, 1953) report two cases in which gastro-ileostomy was accidentally performed. In the first, simple gastro-enterostomy had been performed thirty-six years previously and was discovered only at autopsy. The anastomosis was situated four inches from the caecum, but the pylorus had been patent and allowed the passage of some food through it. In the second case partial gastrectomy had been performed, and thus the situation was more critical. During the seven months between the gastrectomy and the correction of the error in the site of the anastomosis the patient had lost weight and had become apathetic and uncooperative. He had suffered from diarrhœa, his legs were slightly œdematous, and his breath was foul. In addition, there were pronounced hypotension, asthenia and pigmentation, even of the buccal mucosa, all of which suggested some degree of adrenocortical insufficiency. The correct diagnosis of gastro-ileostomy was made on barium meal examination, the condition thus being differentiated from gastro-jejuno-colic fistula, which may cause a similar clinical picture. After the operation four hundred milligrammes of cortisone were given daily for three days with much improvement in the well-being and appetite of the patient, but with little change in the blood pressure. Thereafter his condition steadily improved.

Radical Cancer Surgery.

B. FISHER (*Am. J. Surg.*, February, 1954) reviews the answers received from 25 *questionnaires* on radical cancer surgery sent to prominent surgeons throughout the United States. Supraradical mastectomy (that is, extension of the Halsted procedure to include supraclavicular and internal mammary lymph gland dissections), total gastrectomy for cancer of the stomach, quarterectomy (hind quarter amputation), pelvic evisceration, multiple organ excision and "second look" operations were all considered; but the general feeling was that these procedures were still too new for absolute conclusions, were still in an experimental stage, and were perhaps at present a little disappointing. However, a few surgeons were more optimistic and favoured increased radical surgery, while others believed that although one particular procedure might not be worth while, another one might have great merit.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

CV.

CURRENT THOUGHT ON THE PROPHYLAXIS OF POLIOMYELITIS.

IN the general field of infectious disease there is at the present time more interest in the possible means by which poliomyelitis can be prevented than in any other problem. Opinion is more optimistic than ever before that effective methods will be found, but it should be stressed immediately that the effectiveness of the suggested methods has not yet been established and that it is still too early to decide on what large-scale public health measures should be undertaken. The most hopeful aspect is that, for the first time, the essential features of the problem can be envisaged and the appropriate experiments planned which should eventually supply the answer to the problem of prophylaxis.

To present the situation clearly it is first necessary to outline the natural history of poliomyelitis as it has emerged, especially from studies made in the last ten years. In doing so I shall follow very largely the recent World Health Organization publication on poliomyelitis (World Health Organization, 1954). In any short dogmatic statement of this type there will undoubtedly be points requiring qualification, but there is little likelihood that the main outline will ever need to be modified.

Poliomyelitis, the typical paralytic disease of young children, is the visible expression of a much more widespread process by which various strains of polio virus[1] are transferred from one human being to another. Polio viruses persist in Nature as parasites of the human intestinal tract—there is no evidence that any non-human reservoir of infection exists. Entry into a new host is, in most cases, by the ingestion of material subject to minor fæcal contamination, often by children's fingers. Multiplication of the virus takes place in cells of the tonsillar-pharyngeal region and in the mucosa of the small intestine. From either site, new virus is liberated and is finally excreted in the fæces. Polio viruses are highly resistant to environmental changes, pass readily through the alimentary tract and can survive for long periods in fæcal matter and sewage.

In the great majority of instances infection with the virus produces no symptoms, and the fact of its occurrence can be recognized only by laboratory tests. These show that virus is being excreted in the fæces for a period ranging usually between ten and forty days, and that circulating antibody against the type of infecting virus appears and persists in the blood. If the same type of virus is subsequently ingested, implantation in the alimentary tract does not occur or, if it does, the infection is of much shorter duration.

Symptomatic poliomyelitis is a secondary and essentially accidental result of the primary alimentary tract infection and is mediated by a period of viræmia in which the virus is circulating in the blood. The exact mode by which infection of the central nervous system is initiated is unknown, as are the reasons why the extent to which the central nervous system is involved varies so greatly from one individual to another. These difficulties are, however, not relevant to the current approach to prevention by immunological means, which is directed essentially toward elimination of the viræmic phase.

The most interesting and significant feature of the epidemiology of paralytic poliomyelitis concerns the differences in absolute incidence and in the age distribution of cases, between countries of advanced hygiene and high standards of living on the one hand and primitive unhygienic communities on the other. In Scandinavia, Australia and the United States of America poliomyelitis is a disease of relatively severe incidence, involving especially children of the five to ten years age group, but showing an increasing tendency to produce severe paralysis in young adults. In more primitive, Near Eastern communities it is of a lower general incidence and involves almost exclusively infants (nil to five years). This behaviour is explicable on three assumptions now generally accepted:

1. Strains of polio virus vary widely in the ratio of paralytic-non-paralytic infections produced in susceptible subjects.

2. With the same strain of virus, the likelihood of paralysis in the non-immune subject increases with age at least to young adult life.

3. A non-paralytic infection by a non-invasive strain will provide a high degree of subsequent immunity against paralytic infection by a highly invasive strain of the same immunological type.

Most strains, being relatively non-invasive, will spread freely amongst the infant population of a community having no sense of fæcal hygiene without more than a very occasional paralytic infection in infants. Older children will be thoroughly immune, and even when a highly paralytic strain is introduced, symptomatic disease will be almost limited to the youngest age group. On the other hand, when first contact with a moderately active strain is made, say, at the age of ten years, in an advanced community, paralysis is much more likely to occur.

Attempts to prevent poliomyelitis by immunological means are based on the following facts:

1. There are three immunological types of virus: I (Brunhilde), II (Lansing) and III (Leon). Infection by one type is followed by immunity largely limited to that type.

2. Strains of any serological type may vary widely in their invasiveness for the central nervous system. Severe epidemics are due to the spread of strains of high invasiveness.

3. Post-infection immunity is associated with circulating antibody, a very low concentration of which is sufficient to prevent viræmia after infection by the natural route.

4. As a result of improving fæcal hygiene, the age of first infection with polio viruses is becoming progressively higher and the consequences are becoming correspondingly more severe. It is unthinkable that the enormous benefits that have been derived from improved hygiene in infancy should be discarded, and some acceptable way must be devised of replacing or imitating the process of natural immunization against poliomyelitis during infancy. A method by which infants aged six months to one year can be actively and permanently immunized against paralytic infection is clearly the ideal to be aimed at.

THE PRACTICAL APPROACH TO PREVENTION OF PARALYTIC POLIOMYELITIS.

Quarantine Measures.

Once a satisfactory method of immunization is available, it may become possible to think of eliminating polio viruses completely, but until then we must work on the assumption of their continuing presence. This means that it is desirable for the process of natural immunization to go on at an early age so long as strains of abnormally invasive capacity are absent from the community. In practice, the only way of implementing this is to support the activities of nursery schools and kindergartens in bringing together children from different families at an early pre-school age,

When a severe epidemic like the Victorian one of 1937-1938 appears, however, there is an urgent need to reverse such a policy and to do everything in reason to postpone infection until milder strains are again current. In 1937 there was at least one example of serious spread of paralytic infection in a nursery school, and all such pre-school aggregations of children should be prohibited during a severe epidemic. It will be obvious that there must be both clear judgement and good fortune if an administrator is always to make the correct decision on such points when poliomyelitis reappears in a community.

There is a growing feeling that in the presence of a severe epidemic quarantine methods, particularly if based on laboratory tests for the presence of the virus in contacts, might be more effectively used than they have been in the past. It must always be recognized, however, that, even when the most invasive strains are current, there will be large numbers of children without symptoms carrying and excreting the virus.

The Use of Gamma Globulin.

In animals susceptible to infection by ingestion of polio virus a very low level of circulating antibody in the blood will prevent viræmia and symptomatic infection. It seemed likely, therefore, that administration of concentrated human antibody against poliomyelitis might provide passive protection against paralytic disease. Large pools of normal

[1] In line with a recent recommendation of the committee on virus nomenclature of the International Congress for Microbiology the viruses concerned with poliomyelitis will be referred to as polio viruses.

human blood taken in America contain relatively large amounts of neutralizing antibody against all three serological types of virus. By concentrating the antibody in the γ globulin fraction and injecting a dose corresponding to the amount of antibody in a pint of blood, a significant antibody can be provided for a non-immune child. The antibody level falls according to the normal rules and, for practical purposes, antibody can be regarded as absent six weeks after injection.

In a large and very costly experiment, Hammon *et alii* (1952) showed that children given γ globulin showed significant protection as compared with controls over the period seven to forty days. The indication was that amongst 27,000 children given γ globulin, 38 who would otherwise have had paralytic infections were saved from that fate. Subsequent discussions in the United States have raised some doubts as to the validity of these findings, but my own impression, gained in part from personal discussion with those concerned in the experiment, is that they can be accepted as establishing a significant protective effect from administration of a large dose of γ globulin for a short period of time.

A little thought and calculation will show that it would be economically impossible to adopt passive protection against poliomyelitis by means of γ globulin as a routine public health measure. It is not likely that normal adult human blood contains a great excess of antibody above that needed to ensure protection, so that any practical dose of γ globulin (for example, that corresponding to a pint of blood) will provide a maximal antibody level of only one-tenth or one-twentieth of that of the normal adult, and that level will fall to half the value in ten or fourteen days. To maintain protection of all children at risk for the whole "poliomyelitis season" would require repeated blood donations from every adult in the country.

A further point of importance is that before γ globulin could be used in Australia it would be necessary to show that the pooled serum from which it was prepared did contain adequate amounts of all three antibodies. It is by no means inconceivable that a severe epidemic in Australia might be due to a serological type that had been absent or rare in the country for many years previously and for which adequate antibody was not present in pooled serum from the general adult population.

Opinion as to the best use of the always limited supplies of γ globulin is not yet unanimous. The commonest view is that it should be reserved for family contacts and for other close contacts, as in nursery schools, of paralytic subjects. There is as yet no factual evidence as to its effectiveness in such circumstances. Now that methods for estimating polio antibody are available, γ globulin should not be given to adults whom it is regarded as specially necessary to protect without a preliminary examination of their natural antibody levels. This will, in most cases, show that the γ globulin is unnecessary.

Active Immunization.

There can be no doubt that the ideal prophylaxis would be to provide an active immunity which alone or with the aid of subsequent harmless contacts with active virus would produce a permanent immunity against paralytic infection. No such method has yet been established.

The possibilities currently under study are (i) immunization by formalin-killed tissue culture virus with or without the addition of adjuvants and (ii) administration of attenuated strains of living virus by the alimentary route.

Salk Type Vaccines.

The use of Salk type vaccines is the method currently being tested on a large scale in America. High-titre virus of all three types is grown on tissue cultures of monkey kidney, is inactivated with the smallest safe amount of formalin and, after rigid tests for sterility, is inoculated subcutaneously in three doses of one cubic centimetre each without adjuvant. It is known that this will produce a significant antibody level in inoculated children, and it is hoped that this will provide immunity at least for some years and that occasional "booster" doses in subsequent years will maintain that immunity.

This approach is soundly based and the hopes are eminently reasonable, but it would be most unwise to assume that the Salk vaccine is certain to be effective and free from all danger. That can be shown only by large-scale controlled tests in the field, and only in the United States are there the money, the technical facilities and the driving enthusiasm needed to carry out trials on the necessary scale. The rest of the world must necessarily await the collection of results and their analysis by our American colleagues before deciding whether or not to adopt Salk-type immunization procedures.

At the risk of appearing unduly pessimistic it seems wise to enumerate some of the possible dangers and disadvantages of the Salk method: (i) It is notoriously hard to establish that virus killed by "gentle" methods is completely non-viable, and living virus may persist in some batches of vaccine. (ii) Monkey tissue may be contaminated by known (for example, B virus) or unknown viruses which may be more resistant to formalin that polio virus. (iii) The use of concentrations of formalin adequate to ensure complete non-viability of any viruses present may alter the antigenicity of the virus material and produce ineffective antibody. (iv) Subcutaneous injections of any sort in children incubating poliomyelitis may favour paralytic manifestation of the infection.

Only practical experience can show that none of these are to be feared.

The Use of Living Virus by Mouth.

If the standard interpretation of natural immunity to poliomyelitis is correct, the closest imitation of the natural process would be the immunization of infants by ingestion of strains of each serological type chosen to have no paralytic potentialities. A good deal of attention is being paid to this possibility at the present time, but so far no comprehensive programme to test the method in the field has been worked out. The only human experiments on record by Koprowski and his associates were limited to the use of an attenuated type II strain. As far as they went these experiments were successful. None of the children showed any sign of illness, and in most of them implantation of the virus as shown by its repeated re-isolation from fæces took place. A majority showed signs of active immunization in the form of an antibody response and a resistance to implantation of the same virus given on a second occasion.

Comparable attenuated strains of types I and III have not been tested, but many laboratories are exploring the possibilities of producing such strains by growth of virus in tissue cultures, in the mouse spinal cord, in special lines of mice and in chick embryos. Suitable types may well be available within a few years.

Here, too, there are difficulties and dangers of much the same type as are involved in the use of the Salk vaccine: (i) Attenuation in the last analysis can be tested only in the human subject—a strain safely attenuated for monkeys and most human infants might still be capable of producing paralytic effects in a proportion of unduly susceptible children. (ii) The possibilities of the virus becoming contaminated with another virus would need the same consideration, although the rarity of viruses seriously pathogenic by the alimentary route might make the risk relatively less.

The first of these is the major disadvantage of the approach. It can be put rather differently by saying that the process of developing and testing a living virus vaccine in human subjects would be so haunted by the fear of producing paralysis in the subject that few responsible virologists would care to undertake it. Some, however, may feel strongly enough that the prize of success is worth the risks entailed, especially if preliminary studies are made in subjects under the temporary protection of γ globulin.

Conclusion.

At the present time no adequately based conclusion is possible, and any opinion expressed can represent only a personal synthesis of much reading and extensive discussion with many of those most directly involved in poliomyelitis research. With those reservations I believe that the definitive method of preventing poliomyelitis will be by the use of attenuated living virus, given by mouth to infants in the six to twelve months age group. Such a method is not likely to be developed and accepted within less than ten years. In the interim we must be guided primarily by the results of the current large-scale trial of the Salk vaccine in the United States. If the results show a 50% or better reduction in the incidence of paralysis in inoculated as against control children and there are no unexpected harmful effects of the vaccine, it will be desirable for Australian authorities to develop their own arrangements for providing such protection for those who need or ask for it.

What must be stressed above all is that for the next ten or twenty years immunization against poliomyelitis will be an experimental procedure. It took twenty years' study to bring diphtheria immunization to the stage of a universally

accepted procedure, and the problem of poliomyelitis is at least as complex as that of diphtheria. It will be necessary to have extensive laboratory facilities in any country undertaking the work if mistakes are to be kept to a minimum. Tissue culture methods now make it possible to carry out the two basic procedures of virus isolation and identification and of antibody titration with accuracy and reasonable economy. But it would be very foolish to suggest that the benefit of freedom from poliomyelitis can be had at a cheap rate. Australia will need several teams of epidemiological workers with appropriate laboratory facilities as well as a central laboratory for virus and vaccine production if success is to be won. Much wisdom, too, will be needed to keep Australia effectively abreast of progress in America as well as to embody our own contributions into the major public health crusade of our time—the prevention of poliomyelitis.

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British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney, Crow's Nest, New South Wales, on April 22, 1954. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital.

Infectious Mononucleosis.

DR. F. A. E. LAWES showed a male patient, aged twenty-one years, who had been admitted to hospital on March 12, 1954. Three weeks before his admission he had spent two weeks in a National Service training camp. A few people in the camp were suffering from vague upper respiratory tract infections. Four days before admission he complained of fatigue, listlessness, headache, generalized aches and pains and anorexia. The symptoms persisted until March 11, 1954, when he apparently had a fit. His father said that he heard him cry out and found him in convulsions. From then until his admission he had been delirious, incontinent and very photophobic. On examination the patient was semi-comatose and extremely restless. His temperature was 99·6° F. Neck stiffness and Kernig's sign were present. Lymph glands were palpable in the submandibular region, both groins and both axillæ, but they did not appear to be enlarged. A lumbar puncture was performed at the time of his admission to hospital and revealed clear fluid under a pressure of 150 millimetres. This pressure reading was not accurate because the patient was extremely irritable and throwing himself around the bed. The results of Queckenstedt's test appeared to be positive. The biochemistry of the cerebro-spinal fluid was normal, and the cell count showed 10 red cells, 15 lymphocytes, one monocyte and one polymorphonuclear cell per cubic millimetre. The fluid was sterile. At this time the diagnosis was acute virus encephalitis. As a precautionary measure, intravenous aureomycin therapy was given for twenty-four hours. Within forty-eight hours, the patient had completely recovered, and a blood count and Paul-Bunnell test were performed. The hæmoglobin value was 16·2 grammes *per centum* and the white cell count was 9850 per cubic millimetre. The differential count gave the following information: neutrophile cells 30%, eosinophile cells 2%, basophile cells 1%, lymphocytes 56% (many atypical), monocytes 9%, metamyelocytes 1%, immature cells 1%. The result of a Paul-Bunnell test was positive in a dilution of 1:640. This confirmed the diagnosis of infectious mononucleosis encephalitis.

Patent Ductus Arteriosus.

Dr. Lawes then showed a woman, aged forty-eight years, who over the past three years had complained of occasional fainting attacks, and twelve months before the present time had been told she was hypertensive. Over the past twelve months she had complained of slight breathlessness on exertion. In October, 1953, she developed a throbbing retrosternal pain with an associated feeling of coldness. At the last attack on March 3, 1954, the pain persisted until next morning and was relieved by an injection of morphine. At that time she was admitted to hospital for investigation. On examination of the patient the blood pressure was 140 millimetres of mercury, systolic, and 70 millimetres, diastolic; the heart was not clinically enlarged; there was a continuous murmur maximal at the pulmonary area which was accompanied by a thrill. Electrocardiograms taken at rest were normal, and an electrocardiogram taken after severe exertion showed slight depression of the *T* waves in the precordial leads. Screening of the heart showed that the pulmonary artery pulsations were more prominent than usual; otherwise the cardio-vascular outline was within normal limits. Barium meal examination showed no abnormality of the œsophagus, stomach or duodenum. Dr. Lawes said that in his opinion the patient was suffering from uncomplicated patent *ductus arteriosus*, probably with a narrow lumen; the chest pain was almost certainly not cardiac in origin, and surgery was not indicated. He had not found the cause of the chest pain.

Acute Meningococcal Septicæmia.

DR. F. H. HALES WILSON showed a boy, aged fifteen years, who on the evening before his admission to hospital did not feel well and did not eat his evening meal. At midnight he had an attack of vomiting followed by several other attacks accompanied by diarrhœa. He then became drowsy, confused and doubly incontinent. On examination at 11 a.m. he would respond only to painful stimuli. His temperature was 104° F., his pulse rate 132 per minute, and his blood pressure was 75 millimetres of mercury, systolic, and 45 millimetres, diastolic. There were several small petechial spots on his abdomen and on his scleræ. There were no other abnormal signs on physical examination. A blood count showed 15·2 grammes *per centum* of hæmoglobin and 8350 white cells per cubic millimetre (51% neutrophile cells, 16% lymphocytes, 11% monocytes, 15% band forms, 4% metamyelocytes, 1% myelocytes, 2% blast cells). The erythrocyte sedimentation rate was normal. He was considered to be suffering from septicæmia and was treated with aureomycin, given orally and intravenously, and large intramuscular doses of penicillin. Tube feeding was commenced. During that day his condition deteriorated, and by 6 p.m. there were rapidly spreading giant petechiæ. His pupils were fixed and dilated, and his blood pressure was 48 millimetres of mercury, systolic. He was cyanosed, and neck stiffness was present. He was now diagnosed as suffering from meningococcal septicæmia, and sulphonamides were added to his treatment. From blood taken during the day the meningococcus subsequently grew. His condition during the night slowly improved, and by January 19 all signs had gone except slight general weakness and a persistent pyrexia of 102° F. to 103° F. This pyrexia was not influenced by sulphonamides or by other antibiotics. X-ray examination of the chest, repeated examinations of the urine and repeated blood culture showed no sign of infection. The blood picture, which had shown pronounced leucocytosis, returned to normal. The results of agglutination tests against the common infecting organisms were negative, and the result of the Paul-Bunnell test was negative. Lumbar puncture produced fluid within normal pressure limits. The patient appeared so well that it was felt that the persisting fever must be due to a disturbance of his heat-regulating mechanism, and no further treatment was given. Since then he had steadily improved, had gained weight and was back at school.

Severe Hypertension.

Dr. Wilson then showed a man, aged forty-eight years, who in 1950 had begun to experience severe postural headaches, sometimes associated with vomiting. In 1951 he had become aware of dyspnœa of effort, which steadily increased. Also in this year he had his first attack of acute pulmonary œdema. In 1952 he was no longer able to sleep lying flat, and in 1953 he noticed that his ankles were swollen and that he was tender in the right hypochondrium. During the period from 1952 until his admission to the Royal North Shore Hospital of Sydney, in spite of treatment with digitoxin, aminophylline and phenobarbitone, he had been admitted to hospital on five occasions with pulmonary œdema and with congestive heart failure. Blood pressure readings taken during this period ranged from 250+ to 210 millimetres of mercury, systolic, and 180+ to 130 millimetres of mercury, diastolic. When he was examined on January 26, 1954, he was slightly dyspnœic. His pulse rate was 100 per minute and his blood pressure was 235 millimetres of mercury, systolic, with alternation to 190 millimetres, and 150 millimetres, diastolic. His apex beat was in the sixth intercostal

space five and a half inches from the mid-line. No murmurs were present. He was given "Serpasil" 0·25 milligramme four times daily, and when examined on January 30 his pulse rate was 72 per minute and his blood pressure 195 millimetres of mercury, systolic, and 118 millimetres, diastolic. Alternation was present to 190 millimetres.

On his admission to hospital on the same day his blood pressure was 220 millimetres of mercury, systolic, and 140 millimetres, diastolic, and his pulse rate 76 per minute. His blood urea content was 66 milligrammes *per centum*. An X-ray examination of the chest revealed that the heart was considerably enlarged to the right and left. There was a small effusion at the left lung base and possibly also at the right lung base. The upper lobe of the right lung was shrunken, and there were fibrosis and calcification. An electrocardiogram was consistent with left ventricular hypertrophy. No acid-fast bacilli were found on deposit from the gastric aspirate, and there had been no growth on culture after five weeks. He was treated with digoxin, 0·25 milligramme, three times a day, "Serpasil" 0·25 milligramme, four times a day, and a salt-free diet. He received two injections of a mercurial diuretic with little effect, and administration was then suspended. He did not rest in bed, but was active in the ward throughout the day. His blood pressure steadily fell and during March was consistently within the range 190 to 160 millimetres of mercury, systolic, and 100 to 70 millimetres of mercury, diastolic. He now had no signs of heart failure and was not dyspnœic after walking about 100 yards up a moderate incline. The dose of "Serpasil", which had been increased to three milligrammes a day, was now reduced to one milligramme a day. X-ray examination of the chest showed a small but definite diminution in heart size, and his blood urea content was 59 milligrammes *per centum*. An electrocardiogram showed no significant alteration. He was discharged from hospital to continue to take this dose of "Serpasil" and was examined again on April 2, 1954. He had walked one and a half miles from his home to the hospital and was not dyspnœic. His blood pressure immediately on arrival was 190 millimetres of mercury, systolic, and 95 millimetres, diastolic; after ten minutes' rest it was 180 millimetres of mercury, systolic, and 85 millimetres, diastolic. He felt well in every way.

Mitral Stenosis.

Dr. Wilson's third patient was a married woman, aged forty-three years, who in 1925 had had rheumatic fever, and in 1929 scarlet fever and diphtheria, followed by nephritis. From that time she was relatively well until 1936; then when she was three months pregnant she had an hæmoptysis, but no further symptoms occurred at the time. In 1939 she had a Cæsarean section for reasons other than her cardiac condition. In 1940 she noticed that she tired easily and became breathless on mild exertion. This continued until 1946, when she had frequent severe attacks of breathlessness associated with hæmoptyses; a pregnancy at that time was terminated, and she was sterilized. From 1947 to 1952 she was reasonably well, however, except for occasional attacks of breathlessness associated with substernal pain. In November, 1952, and again in December, 1952, she had attacks of paroxysmal tachycardia, the first lasting about four hours and the second about half an hour. Towards the end of April, 1953, she had another attack of paroxysmal tachycardia; that abated on the same day, but recurred on the next two days; the last attack persisted for three days until her admission to hospital and was associated with substernal pain.

On examination of the patient her pulse rate was 140 per minute, the pulse being quite regular in time and amplitude, there was no obvious cardiac enlargement, and a harsh diastolic murmur was audible at the mitral area. The rapid pulse was not affected by carotid sinus or eyeball pressure, and an electrocardiogram at that stage showed probable nodal tachycardia. Digitalization was commenced and the rhythm converted immediately to that of an auricular fibrillation; at the same time she had an episode of cerebral embolism, resulting in left hemiparesis, which fortunately resolved in forty-eight hours. Anticoagulant therapy was commenced and carried on for one week. Further investigations were then proceeded with as it was considered that the patient was a candidate for mitral valvotomy. The findings on X-ray examination of the chest and screening were consistent with pure mitral stenosis, and there was only a small degree of calcification visible in the mitral valve. An electrocardiogram showed fibrillation. Her hæmoglobin value was 15·7 grammes *per centum*; leucocytes numbered 19,900 per cubic millimetre and were of normal distribution. The blood sedimentation rate was 11 millimetres per hour. Digoxin therapy was continued throughout this period, being given in alternate doses of 0·25 milligramme twice daily and three times a day. She appeared to be intolerant to any larger doses.

Mitral valvotomy was performed by Dr. Ian Monk on June 3, 1953, when it was found that the pulmonary artery was about twice the normal size; there was a large left auricle; the orifice of the mitral valve was between five and 10 millimetres in diameter, the posterior cusp being thickened and possibly calcified, and less mobile than the anterior. The lateral commissure was split with the finger, and the valve at the end of the operation was estimated as two to three fingers in diameter. Post-operatively she was given continuous oxygen therapy in an oxygen tent for forty-eight hours and required large doses of digitalis, up to six tablets daily for this period, because of a rapidly increasing pulse rate. She was then able to take her previous dosage of digoxin with fairly adequate control; the fibrillation, however, persisted. Examination two weeks later revealed auricular fibrillation, persistent "opening snap" and a very soft diastolic mitral murmur. It was considered that the result of the operation was good, and it would be advisable to try to convert her back to normal rhythm. Anticoagulant therapy was commenced and continued for one week, when quinidine, grains six two-hourly, was commenced, and after three doses of this her rhythm had reverted to normal. Digoxin therapy was suspended; a maintenance dose of quinidine, grains three four-hourly, was continued for the next forty-eight hours. A few days later auricular fibrillation returned, and after treatment with phenyl indanedione ("Dindevan") to control coagulation another attempt was made to produce normal rhythm by quinidine. This did not succeed. Four months later fibrillation was still present, and as she could not tolerate adequate dosage of digoxin she was readmitted to hospital. This time treatment was successful with quinidine, after preliminary control of coagulation with "Dindevan". Her exercise ability was now much better than before operation ten months previously.

Severe Hypertension with Retinopathy.

Finally Dr. Wilson showed a woman, aged twenty-nine years, who four years previously had been found to have hypertension. She was admitted to hospital in September, 1953, for investigation and treatment. On that occasion her blood pressure was 255 millimetres of mercury, systolic, and 170 millimetres, diastolic, on admission and did not fall below 250 millimetres of mercury, systolic, and 160 millimetres, diastolic, without treatment. There was severe hypertensive retinopathy, and in the urea concentration test the maximum specific gravity was 1·017 and the urea concentration 2·9%. The urine contained no albumin. No evidence of coarctation of the aorta, phæochromocytoma or unilateral renal disease was found, and no relevant family history was elicited. She was treated with "Veriloid" (extract of *Veratrum viride*), "Hydralazine", "Serpasil" Ciba (reserpine) and "Apresoline" Ciba, and was on a low salt diet. After gradual increase the final dosage was "Veriloid" eight milligrammes per day, "Serpasil" 0·75 milligramme per day, and "Apresoline" 200 milligrammes per day.

She was readmitted to hospital for reassessment of her renal function, and during her stay in hospital from February 19 to March 6, 1954, the systolic pressure remained within the range 160 to 130 millimetres of mercury and the diastolic within the range 110 to 90 millimetres. The blood urea content was 31 milligrammes *per centum* and the urea clearance 46% of average normal. The urea concentration test showed a maximum concentration of 2·7% in a volume of 48 millilitres. The maximum specific gravity during the test was 1·014 and at any time was 1·018. She was examined in consultation by Dr. K. B. Armstrong, who reported that there were neither hæmorrhages or exudates in either fundus, and that the spasm of the main retinal arteries was much less. She felt well in herself and complained of only slight blurring of vision for distant objects. Other investigations revealed the presence of a duodenal ulcer. She was discharged from the hospital to continue with the same therapy as before.

Tubular Rickets, Hereditary Blue Sclerotics and Facial Hirsuties.

DR. DOUGLAS ANDERSON showed a girl, aged two years, whose legs had become very bowed at the age of twelve months, when she started to walk, notwithstanding that she received at least 1500 units of vitamin *D* every day. Otherwise she was a fine-looking child. X-ray examination of the knees showed rickets. At the age of twenty months, after treatment, the serum calcium and inorganic phosphorus values were found to be normal; the value for the serum

alkaline phosphatase was still abnormally high, but falling. Her urine was always alkaline when passed. It was thought that the rickets was due to a renal tubular defect. Dr. Anderson suggested the following mnemonic for the treatment of Dent's six types of tubular rickets:

One, two and three,
Vitamin *D*;
Four, five and six,
Citrate will fix.

He said that the child had done very well on 50,000 units of vitamin *D* daily, so that it was not proposed to facilitate the absorption of calcium from the gut with citric acid or the calcifiability of the bones with citrate. But it was proposed to keep her under observation, so that the optimum dose of the vitamin would not be exceeded. The child was observed to have blue sclerotics, as had her mother also. At the age of twenty months it was noticed that she was growing hair on the sides of the face ("side levers"), and her 17-ketosteroid excretion was found to be three times the normal value for her age.

Obstinate Cardiac Œdema Treated without Salt Restriction.

Dr. Douglas Anderson also showed a man, aged sixty years, suffering from congestive heart failure, in whom, prior to admission to the hospital, œdema had persisted despite treatment with digitalis and mersalyl and a low-salt diet. He had been weak and cyanotic and mentally a little dull, presumably because the mersalyl given in conjunction with a low-salt diet had produced hypochloræmia. His tongue had been dryish, and his face had looked pinched, because hæmoconcentration and cellular dehydration had been allowed to develop. He was given four pints of liquid daily to facilitate diuresis and the excretion of sodium by the kidney; also an acid-ash diet and acidifying drugs, so that sodium bicarbonate would be mobilized from the œdema fluid to correct the tendency to acidæmia. He was allowed a normal amount of dietary salt, so that he would receive enough for physiological needs. A low-salt diet would have been given only if the kidneys had not been able to excrete sodium readily.

Hypertension.

DR. R. D. PUFLETT showed five patients suffering from hypertension.

The first patient had had two attacks of severe left ventricular failure secondary to essential hypertension with a blood pressure of 190 millimetres of mercury, systolic, and 130 millimetres, diastolic. The retinal arteries were normal. An enlarged heart with presystolic gallop rhythm was present. An electrocardiogram showed left heart strain. Each attack was dramatically controlled with parenteral hexamethonium bromide therapy, and the patient became normotensive. However, after a few months his blood pressure began to rise and was now 170 millimetres of mercury, systolic, and 100 millimetres, diastolic. He was receiving "Serpasil" one milligramme daily.

The second patient was a woman, aged sixty-four years. In September, 1951, after she had been under several years' observation for essential hypertension, her blood pressure rose to 220 millimetres of mercury, systolic, and 160 millimetres, diastolic; very extensive exudates and hæmorrhages appeared in both eyes, with papilloedema. Albuminuria was present and cardiac enlargement with presystolic gallop rhythm. An electrocardiogram showed left heart strain. She was given hexamethonium bromide parenterally with a dramatic lowering of blood pressure. Treatment was then changed to "Vegolysin" given orally, and now she was having "Ansolysin", 1200 milligrammes daily, and "Serpasil", 0·75 milligramme daily. The blood pressure was now reduced to 160 to 190 millimetres of mercury, systolic, and 100 to 110 millimetres of mercury, diastolic. Albuminuria had disappeared. The exudates and hæmorrhages had subsided and were greatly reduced in size; and over the last twelve months they had been static and were now regarded as residual scarring. Dr. Puflett said that the case was a great triumph for the combination of "Ansolysin" and "Serpasil".

The third patient had presented with congestive cardiac failure. The blood pressure was 220 millimetres of mercury, systolic, and 120 millimetres, diastolic. The retinal arteries were narrowed, and small hæmorrhages were present. "Serpasil", one milligramme daily, was given and controlled the blood pressure to 170 millimetres of mercury, systolic, and 80 millimetres, diastolic.

The fourth patient had been admitted to hospital with a blood pressure of 210 millimetres of mercury, systolic, and 140 millimetres, diastolic. "Vegolysin" reduced it to 170 millimetres of mercury, systolic, and 100 millimetres, diastolic. With "Ansolysin", 600 milligrammes daily, and "Serpasil", two milligrammes daily, it had now decreased to 170 to 180 millimetres of mercury, systolic, and 110 to 120 millimetres of mercury, diastolic.

The last patient of the group had presented with congestive cardiac failure, and a blood pressure of 200 millimetres of mercury, systolic, and 130 millimetres, diastolic. The retinal arteries were irregular and narrowed. Albuminuria was present. "Ansolysin" lowered the blood pressure to 160 millimetres of mercury, systolic, and 100 millimetres, diastolic, and the albuminuria cleared up. The blood pressure was being maintained at the lower level with "Serpasil" only, 0·75 milligramme daily. Satisfactory control was first effected with moderate doses of "Ansolysin" and was being satisfactorily carried on with "Serpasil".

Dermatomyositis.

Dr. Puflett then showed a man, aged forty-eight years, who in May, 1953, had noticed pains in the muscles of the thighs, forearms and calves, with numbness of hands and feet. A few weeks later swelling appeared in the painful muscles. The muscles were tender and had pitting œdema. Atrophic changes in the skin followed, and the muscles became indurated. On examination the muscles of the extremities were hard, and the overlying skin had become atrophic and immobile. These changes were greater at the periphery of the limbs. Biopsy showed the condition to be dermatomyositis. The patient had been receiving cortisone, which he maintained made him less rigid. It had not affected the condition, which appeared to be slowly spreading up the limb.

Clonorchis Sinensis.

Dr. Puflett showed another patient, a man, aged twenty-two years, with a history of recurrent diarrhœa and persistent eosinophilia. *Clonorchis sinensis* ova were found in his stools. Dr. Puflett said that *Clonorchis sinensis*, a fluke, was endemic to the Far East. The intermediate hosts were freshwater fish. The fluke developed from cysts in the infested fish, and involved the bile ducts, causing a primary proliferation of the biliary epithelium and finally cirrhosis.

Dystrophia Myotonica.

The next patient shown by Dr. Puflett was a married woman, aged thirty years. Her history was that for an indefinite period of time she had been unduly weary and easily fatigued. She noticed that she could not turn her eyes to the side. A swelling was present in the lower part of the neck. Her menstrual periods were normal. She had had diphtheria, and had undergone tonsillectomy in childhood. It had not been possible to obtain a family history. On examination, the patient was noted to be a thin, prematurely aged woman with a nasal toneless voice and bilateral complete external ophthalmoplegia. She had wasting and weakness of the facial muscles and of the muscles of the upper extremities, shoulder girdle, neck (particularly the sterno-mastoid), left thigh and back. There was myotonia of muscles of the forearms and hand and the myotonic reflex was present. She had a soft diffuse swelling of the thyroid gland. An electrocardiogram showed myocardial impairment with inverted *T* waves.

Coarctation of the Aorta.

Dr. Puflett's last patient was a man suffering from coarctation of the aorta. He had been symptom-free, but at a routine medical examination had been told that his heart was abnormal. Examination of the patient showed diminished *dorsalis pedis* and posterior tibial artery pulsations. The femoral arterial pulsation was palpable but lagged noticeably behind the radial. Arterial pulsation could be felt along the medial border of the left scapula and down the internal mammary arteries. The brachial artery blood pressure was 170 millimetres of mercury, systolic, and 100 millimetres, diastolic, in both arms. The apex beat was normal in situation. A moderately loud systolic murmur could be heard best between the sternum and apex, radiating into the axilla, and soft blowing systolic and diastolic murmurs could be heard at the aortic area. Screening examination showed an enlarged left ventricle. Surgical treatment had been recommended.

(To be continued.)

NEW SOUTH WALES BRANCH NEWS.

Annual Branch Prize for an Essay on a Scientific Subject.

THE following are the conditions applicable to the Annual Branch Prize established by the Council of the New South Wales Branch of the British Medical Association for an essay on a scientific subject:

1. The Council of the New South Wales Branch of the British Medical Association will each year award a prize of 100 guineas and a bronze medallion for an essay on a scientific subject.

2. Entries for the prize competition will be received from any ordinary member of the Branch. Entries will close at midday on November 30, and the prize will be awarded to the successful essayist at the succeeding annual meeting.

3. Essays will be on a subject chosen for the purpose and should not be longer than 12,000 words. The use of illustrations and graphs will be permitted. Where necessary, complete references should be given.

4. Work that has already been published will not be acceptable, but no objection will be raised if a small preliminary communication to establish priority has been accepted for publication by or has been published in a reputable medical journal.

5. Intending competitors shall notify the Medical Secretary of the Branch of their intention to submit contributions at least two months before the closing date.

6. Two or more examiners shall be appointed each year.

7. The winning essay shall become the property of the Branch Council, which will submit it for publication in an appropriate journal. If in the opinion of the judges no essay is of sufficient merit, no award shall be made.

8. Three copies of an essay shall be submitted. Essays are to be typewritten and double-spaced lines are to be used. Each essay shall bear a motto or *nom de plume*, and shall be accompanied by a sealed envelope containing the author's name and qualifications.

9. The Organization and Science Committee of the Branch Council shall be the committee controlling the award.

The subject chosen for the year 1954 is: "The Present-Day Pattern of Infectious Diseases."

Entries close at midday on November 30, 1954.

Notification of intention to submit contributions must be forwarded to the Medical Secretary, British Medical Association, New South Wales Branch, 135 Macquarie Street, Sydney, not later than September 30, 1954.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

AMPUTATION OF A LEG IN RIO DE JANEIRO.[1]

[From White's "Journal of a Voyage to New South Wales".]

September, 1787.

WHILE we remained here,[2] the weather being cool and favourable I prevailed on the surgeon who was about to amputate a limb to allow me to take it off according to Allenson's method. During the operation I could plainly see that he and his pupils did not seem much pleased with it; and he told me afterwards it was impossible it could ever answer. A very short space of time however made them of a very different opinion; and in eighteen days after, when we sailed I had the satisfaction to leave the patient with his stump nearly cicatrized, to the no small joy of the surgeon, who said, that if the man had died, he should have been heavily censured for making him the subject of experiments. The circumstance of a man's leg being cut off and almost healed in as many days as it generally takes weeks soon became known, and added very much to the estimation in which the people of this place held English surgeons. Whenever I visited the hospital afterwards, the objects of pity with which it was filled, used to crowd round me in such a manner and in such numbers, for my advice that I found it difficult to get from them. And they now would readily have submitted to any operation I should have proposed, but as I saw the surgeon did not much approve of my interference I gave up all ideas of it.

[1] From the original in the Mitchell Library, Sydney.

[2] Rio de Janeiro.

Correspondence.

THE TRAINING OF MEDICAL STUDENTS.

SIR: I wish to comment on some of the points in the letters of Thomas F. Rose and Ellen M. Kent Hughes in the journal of July 10.

It would appear that Dr. Rose is out of touch with developments in medical education, as there are many reports on dissatisfaction with undergraduate medical education, not only here but in Great Britain and in America.

"It is as easy to lecture to 200 students as to 6." The same is true of 600 students, which was the size of the class some years ago in biology here in Queensland. When this state of affairs is allowed much is lost of the relationship that should exist between lecturer and student—no lecturer (except be he a very exceptional man) may expect to retain a high standard when he has to give lectures, say, three times a week for thirty weeks.

"Nowadays, too, teaching is more efficient and concentrated than heretofore." I think this statement is open to question. There were quite a number of first-class lecturers when I did my course over forty years ago, among them Professor Welsh in pathology, Sir Herbert Maitland in clinical surgery and Dr. Sydney Jamieson in clinical medicine. I have listened to many lecturers since, both in England and in Australia, and these three of our lecturers were equal to the best.

"One can remember times when a group of students would wait in vain for hours for an honorary who did not appear." This was certainly true, and this practice urgently called for reform, but to expect a registrar to take the place of an honorary is no solution of the problem. I am of the opinion that the advice of the Editor of THE MEDICAL JOURNAL OF AUSTRALIA should be followed, and a conference held of the profession including all members interested in medical education. Up to the present little interest in this subject seems to have been taken by our professional leaders.

"The whole outlook and aim of undergraduate medical education have changed. . . ." I would commend to Dr. Rose the article on undergraduate medical education in the *British Medical Journal*, May 15, 1954.

"The University now aims to produce doctors with a sound general medical education (necessarily mostly theoretical). . . ." In the last three words lies the reason for the "present discontent". The truth of the matter is that not only have we to take into consideration the statement by an English authority that the "exaltation of theory to the detriment of practice is the hall-mark of academic decay", but we have as a practical measure to decide on the relative claims of undergraduate and graduate teaching. This problem is all the more urgent as all graduates (at any rate in England) are expected to spend one year resident in hospital.

Dr. Kent Hughes has stated that she finds graduates are not well trained in the everyday affairs of general practice. I can vouch for this, as many graduates have told me that they get insufficient experience in anæsthetics, midwifery, pædiatrics and psychiatry during their twelve months' term as resident medical officers.

Nor is all well with graduate training either in medicine or surgery. I am informed (though I write subject to correction) that the regulations for the Fellowship of the Royal Australasian College of Surgeons have been greatly reduced. I refer particularly to those regulations that provided for apprenticeship with a recognized surgeon.

In the same way there should be apprenticeship for those who are taking the Membership of The Royal Australasian College of Physicians. (Many of the graduates who qualify for these degrees have not been outside a hospital and have not taken full responsibility for major work.) We have the

situation where young graduates have the academic knowledge, but insufficient experience. On the other hand there are many older practitioners qualified by experience, but without the academic knowledge, other than that gained prior to graduation, and yet these graduates are not allowed to call themselves specialists in Queensland.

It should be remembered that there are many qualities of hand, heart and mind required of specialists to make them good doctors, and these do not lend themselves to examination.

Would we not do better to adopt the European and North American system of granting an M.D. on graduation and after that prescribing a period of years for training as a specialist?

There were many very able Australian graduates of the last part of the nineteenth century and part of the twentieth who were second to none as general practitioners—the ranks of the specialists were largely recruited from such as these. At the same time, many graduates could get no hospital experience, and I think a compulsory year in hospital is one of the greatest advances we have made in medical education. However, I think the hospitals should give more attention to training these graduates for general practice, for at the present time the services of many of them are only being considered in relation to the hospital's needs.

Other points, too, in graduate and undergraduate training are that some means should be found to overcome the difficulty of providing training owing to the very many specialties that now exist, and for more attention to be given to training beyond the boundaries of the hospital where some 65% of graduates are destined to carry on their life's work.

Yours, etc.,

E. S. MEYERS,
Acting Professor of Social and Tropical Medicine.

University of Queensland Medical School,
Brisbane,
July 13, 1954.

PEPTIC ULCER.

SIR: It must be somewhat unusual for a doctor to have the opportunity of observing a patient with peptic ulcer over a period of some forty years. I have—the patient—myself. I know your readers could not bear to read a detailed history, but suffice it to say that the ulcer is duodenal, has been radiologically verified and has put me in hospital with two massive hæmorrhages, one twenty-eight, the other five years ago.

After reading extensively on the subject and having followed many of the regimes and remedies recommended, I can only say that my ulcer gets greater and quicker relief from a little milk than from anything else. Milk cannot always be carried about when away from home, but a few antacid tablets are a comfort to have in one's pocket. I still think that bismuth, with or without belladonna, taken before meals, is equal to or superior to most of the boosted fancy remedies on the market.

Finally, sir, none of the theories in vogue have helped me to discover why I can be quite comfortable for months at a time, nor why one day that stealthy pain begins to creep across the epigastrium again, nor even less why it suddenly disappears again for another remission.

Yours, etc.,

A. W. J. BULTEAU.

143 Macquarie Street,
Sydney,
July 12, 1954.

A SIGNAL HONOUR FOR TWO AUSTRALIANS.

SIR: There has just been published in the United States of America a book entitled "Classics in Clinical Dermatology", which should I feel be brought to the attention of your readers and yourself. Walter B. Shelley, Chief of the Clinic of Dermatology, University of Pennsylvania, and John T. Crissey, themselves both outstanding investigators, have collected for the first time the original descriptions of skin disease entities. They point out that "the establishment of a disease picture requires of the observer the power to perceive the new as the new, and although ninety-five men whose descriptions are presented differed in every conceivable way—race, age, time, background, education, temperament and ability—they had this one faculty in common".

The collection starts with the description of psoriasis by Robert Willan in his "Treatise on Cutaneous Disease" (1808). It contains such famous names, to list but a few, as Bazin, Addison, Jonathan Hutchison, Unna, Boeck, Quincke, Castellani and many others such. The final classic listed, sir, you will be pleased to know is "Tropical Anidrotic Asthenia: A Preliminary Report", published in THE MEDICAL JOURNAL OF AUSTRALIA in 1944 by Stuart D. Allen and John P. O'Brien.

Though Allen has retired from practice, O'Brien has made further original contributions to our knowledge of diseases associated with sweating and sweat glands and the problems of staphylococcal infection of the skin so important in every phase of medicine and surgery. This work, strangely enough, has been appreciated more and is far better known in America than here. Indeed Sulzberger, the editor of "The Year Book of Dermatology", was moved to comment: "J. P. O'Brien is one of the most brilliant, fertile and indefatigable among the modern investigators of the local and systemic effects of poral closure and the sweat retention syndrome."

It is to be hoped, sir, that when the plans of the National Health Service dealing with research in disease come to being, use will be made of men of this calibre, who have already achieved so much with facilities and help that would make the most junior worker in America turn away in disdain.

Yours, etc.,

ADRIAN JOHNSON,
Editor, *Australian Journal of Dermatology*.

185 Macquarie Street,
Sydney,
Undated.

References.

ALLEN, STUART D., and O'BRIEN, J. P. (1944), "Tropical Anidrosis: Tropical Anidrotic Asthenia", M. J. AUSTRALIA, 2: 335.

SHELLEY, WALTER B., and CRISSEY, JOHN T. (1954), "Classics in Clinical Dermatology", Charles C. Thomas, Springfield, Illinois.

SULZBERGER, M. B., and HERMAN, F. (1950), "The Ætiology of Poral Closure", discussion, *J. Invest. Dermat.*, 15: 53.

RUPTURED PEPTIC ULCER OR DIABETES MELLITUS?

SIR: The case reported in your journal of June 5 takes me back to one almost identical about which I sent a letter to the *British Medical Journal*. This was printed in the journal of October 8, 1949, under the title "Acute Abdomen in Diabetes Mellitus", and, like your contributor, I asked for explanations of the condition.

In the literature, various mentions have been made of this occurrence. In my letter I made reference to the "acute abdomen" of ketosis (1948) and mention of it by Zachary Cope (1946). When I was confronted with my case I was aware of the existence of such a condition. In all respects, the present case appears to be identical with mine. Regarding his pain, I quote from my letter of 1949: "He was writhing in agony with abdominal pain and continuously crying out to be given something to relieve his pain." Like the present case my man had vomited at the onset and was sure the vomiting had preceded the pain. This fact is mentioned by Lawrence (1950) as important in the differential diagnosis.

In my case the man was a known diabetic, and the onset of the pain had occurred eighteen hours prior to my seeing him. Also I had the history that for three days he had not had any insulin. What influenced me to withhold surgery was the fact that, despite the picture of an acute abdominal catastrophe, the signs were not in keeping with an acute abdomen of eighteen hours' duration. Having made this decision, morphine was administered, and he settled quickly on an intravenous saline drip, through which treatment of the hyperglycæmia was given.

In response to my letter in 1949 Dr. Elliott P. Joslin, of Boston, communicated to me the substance of an article he was about to publish. This later appeared in the *Review of Gastroenterology* in August, 1950. According to him, our cases would be either due to gastric atony or pseudo-peritonitis and may be linked with sodium chloride depletion.

McKittrick (1933) has seen and studied many instances of this type of case. He states that in all cases operated upon with negative findings, a diagnosis of an extensive

surgical lesion, such as acute pancreatitis, general peritonitis or perforated ulcer, has been made. He also points out that, just as severe abdominal pain may be the outstanding symptom of a patient with impending coma, it is the first symptom to disappear under proper insulin treatment. He accepts malaise, drowsiness, vomiting and diffuse abdominal pain with widespread tenderness and spasm in a diabetic, as evidence of acidosis. With such cases he suggests that operation should not be performed unless the abdominal symptoms persist after three or four hours of adequate insulin treatment. Conversely, however, he states that a history of abdominal pain with or without vomiting, when associated with localized and definite abdominal tenderness, usually with spasm, is suggestive of a surgical intra-abdominal lesion in patients with diabetic acidosis, just as it is in a non-diabetic patient, and may be the indication for immediate operation. He states that wherever definite differentiation is impossible and yet imperative, it may be safer to open the abdomen than to suffer further delay. Beardswood (1935) has discussed in detail the abdominal symptomatology of diabetic acidosis after a study of a thousand cases. Birch (1948) suggests that the acute abdominal pain may be due to distension of the liver by fat, but I know of no proof of this theory. Young (1951) mentions this condition and, like other authorities, advises active treatment of the dehydration and ketosis as an initial step in treatment. He stresses, however, that should such measures fail to produce a rapid response in the abdominal condition, laparotomy should be undertaken.

The cause of this condition is difficult to discover. That a ketosis exists due to insufficient carbohydrate metabolism from lack of insulin is accepted. The ketone bodies by themselves are no longer regarded as poisons and can be used efficiently by the body as a source of energy in the ketosis of diabetes, in fasting conditions and in normal metabolism. The lowered serum potassium has not been considered a possible cause. It is likely that the factors of dehydration and chloride depletion are in some way the cause of the condition, though the exact way in which they do so is still not understood.

Yours, etc.,

187 Macquarie Street,
Sydney,
June 29, 1954.

M. GLICK.

References.

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LAWRENCE, R. D. (1950), "The Diabetic Life", Churchill, London, 30.
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McKITTRICK, L. S. (1933), "Abdominal Symptoms with or without Abdominal Lesions in Diabetic Acidosis", *New England J. Med.*, 209: 1033.
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YOUNG, B. A. (1951), "Ketosis and Coma in Diabetes Mellitus", *Postgrad. Med. J.*, 27: 309, 338.

A NOTE ON BRONCHIECTASIS.

SIR: My reading of Dr. McManis's letter in your journal of July 3, 1954, leads me to conclude with regret that he has missed the import of my short article. In this letter there is intense preoccupation with the claim that my article contains nothing to prove that infective changes in the lung without bronchial dilatation can produce symptoms of bronchiectasis. The submission of my article is simply that in juvenile "clinical bronchiectasis", as defined, dilated bronchi are not coextensive with diseased lung tissue, and hence bronchography cannot be relied upon to define the extent of the disease.

Bronchography has been very largely used with the object of defining the extent of diseased lung and has been accepted as decisive in determining whether a lingula is to be removed at the same time as a left lower lobe or a middle lobe at the same time as a right lower lobe. Series of cases handled along these lines must therefore exist and be available for post-operative follow-up study.

The suggestion implied in my article is that those with records of such series at their disposal may be interested to ascertain if the proportion of cases not cured by operation is comparable to the proportion that would be predicted

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 10, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	4(3)	3(3)	2(2)	..	..	..	..	..	9
Amœbiasis	..	..	..	..	1(1)	..	..	..	1
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1	..	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	6(3)	10(10)	1	..	..	..	..	..	17
Diphtheria	5(3)	4(4)	3(2)	..	6(4)	..	1	..	19
Dysentery (Bacillary)	..	1(1)	2(2)	..	..	..	..	..	3
Encephalitis	..	1	..	2(1)	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	2	..	..	..	1	..	..	3
Infective Hepatitis	32(10)	6(2)	..	..	1	..	..	1	40
Lead Poisoning	..	..	4	..	..	..	..	..	4
Leprosy	..	..	..	..	12	..	..	..	12
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	1(1)	2(1)	..	1(1)	..	..	..	4
Meningococcal Infection	6(6)	1	..	2(2)	1(1)	..	..	..	10
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	7(2)	10(5)	1	8(7)	5(1)	..	..	..	31
Puerperal Fever	1	..	..	..	..	..	..	..	1
Rubella	..	4(2)	..	..	8(7)	..	..	..	12
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	20(15)	14(9)	15(11)	4(1)	2(2)	2	..	..	57
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	2(1)	..	..	..	..	..	2
Trachoma	..	..	..	..	20	..	..	..	20
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	91(85)	33(20)	10(7)	6(5)	7(6)	4(1)	3	..	154
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

from my interpretation of my data, namely, four out of fifteen in the presence of unilateral disease and approximately one out of two in the presence of bilateral disease. Such a study may help to resolve Dr. McManis's perplexities.

Yours, etc.,

The Royal Newcastle Hospital, Newcastle, New South Wales. J. R. S. DOUGLAS.

July 16, 1954.

Notice.

THE ROYAL MELBOURNE HOSPITAL RESIDENT MEDICAL OFFICERS' CLUB.

THE Royal Melbourne Hospital Resident Medical Officers' Club will hold a special Friday lunch-time meeting at 1 p.m. on August 27, 1954, at which Dr. E. E. Dunlop and Dr. I. H. McConchie will discuss the surgery of arterial disease. Sir James Learmonth will be present and will contribute to the discussion. Lunch will be served from 12.30 p.m.

Corrigenda.

THE RUPERT DOWNES MEMORIAL LECTURE.

THE Rupert Downes Memorial Lecture is a foundation of the Royal Australasian College of Surgeons. This fact was unfortunately not stated in the publication of the lecture by Dr. Allan S. Walker in the issue of July 17, 1954.

PHÆOCHROMOCYTOMA CURED BY SURGICAL REMOVAL.

AN error appears in the article "Report of a Case of Phæochromocytoma Cured by Surgical Removal", by R. G. Lewis and L. W. Knight, published in the issue of June 26, 1954, at page 978. In the third paragraph of the article and in the fourth line the phrase "his temperature was 90° F." should read "his temperature was 96° F.".

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Fuller, Kenneth Thomas, M.B., B.S., 1952 (Univ. Sydney), 79 Station Street, Arncliffe, New South Wales.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Bedford, Roma Margherita, M.B., B.S., 1954 (Univ. Sydney); Dawson, Ian Henry Edward (provisionally registered), M.B., B.S., 1954 (Univ. Sydney); Delves-Deffee, Edward Peter, M.B., B.S., 1954 (Univ. Sydney); Hanley, Brian Robert, M.B., B.S., 1954 (Univ. Sydney); Henderson, Bruce Leslie (provisionally registered), M.B., B.S., 1954 (Univ. Sydney); Holland, Victor Wallace, M.B., B.S., 1954 (Univ. Sydney); Murphy, Arthur James O'Grady, M.B., B.S., 1954 (Univ. Sydney); O'Neill, Gerald Allen (provisionally registered), M.B., B.S., 1954 (Univ. Sydney); Posney, Kazimierz, M.B., B.S., 1954 (Univ. Sydney); Redhead, Harris Frank, M.B., B.S., 1954 (Univ. Sydney); Wallace, Mary Helen Katherine (provisionally registered), M.B., B.S., 1954 (Univ. Sydney); Woodforde, John Marcus, M.B., B.S., 1954 (Univ. Sydney); Morrison, James Brown, M.B., B.S., 1953 (Univ. Sydney); Smith, Colin Landon, L.M.S.S.A., M.B., B.S., 1953 (Univ. London); Quoyle, John Charles Joseph, M.B., B.S., 1948 (Univ. Sydney); Roberts, Ivor Charles, M.B., B.S., 1952 (Univ. Adelaide); Ambroza, Sophie, provisionally registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1953; Caska, Zdenko Josef, provisionally registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1953; Derkatsch, Helene, provisionally registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1953; Madirazza, Spasoje, provisionally registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1953; Nickelburg, Tadeusz, provisionally registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1953; Perendi, Serafima, provisionally registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1953.

Deaths.

THE following deaths have been announced:

SHEEHAN.—Paul Alexander Sheehan, on June 19, 1954, at Merewether, New South Wales.

KENNEDY.—John Araluen Kennedy, on July 14, 1954, at Sydney.

Diary for the Month.

AUG. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 4.—Victorian Branch, B.M.A.: Branch Meeting.
AUG. 4.—Western Australian Branch, B.M.A.: Council Meeting.
AUG. 6.—Queensland Branch, B.M.A.: General Meeting.
AUG. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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